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## ON THE ETIOLOGY OF HYDRAMNIOS.

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AMONG the unsettled questions of obstetric medicine the cause of the condition called Hydramnios or Dropsy of the Amnion still holds a place. Thus, Dr. Playfair referring to it writes, "Its precise cause is still a matter of doubt. By some it is referred to inflammation of the amnion itself, at other times it is apparently connected with some morbid state of the decidua." Dr. Leishman says, "It has been supposed to be due to inflammation of the amnion, constitutional syphilis, or to some diseased condition of the foetus; but although all these theories are possible, none of them have up to this period been demonstrated." Caseaux says, "Il est impossible dans l'état actuel de la science de préciser la cause de cette singulière affection." Earlier authors write in the same strain. It has occurred to me that if the source of the liquor amnii can be established it will become less difficult to understand the cause of its occasional excess. The pathology of the disease will be elucidated by a reference to the physiology of the structures involved. Some have held the liquor amnii to be derived from the mother, others from the foetus, and others from both. That it is not, at all events wholly, of embryonic origin may, I think, be held to be proved by the fact, as stated by Schroeder, that in cases where the embryo has become atrophied, or has even entirely disappeared, nevertheless liquor amnii has been present, and in an amount corresponding to the age of the ovum without

reference to the embryo. It contains, doubtless, certain foetal contributions, such as epidermic scales, urinary constituents, and more rarely, and generally only towards the end of gestation, traces of meconium, but all of varying amount, and not of its essence. Essentially it is a limpid serous fluid, of slightly alkaline reaction, containing a trace of albumen and some saline constituents, especially chloride of sodium and phosphate of lime; and only with the progress of gestation does it contain the other matters referred to, urea, thrown-off epidermic scales, lanugo, and meconium. Of these the urea is the most constant and important, and undoubtedly represents a certain amount of renal secretion habitually added to the liquor amnii. That this is so is proved by the occasional occurrence of congenital hydronephrosis of one or both kidneys by obstruction in some part of the urinary system. The theory that the liquor amnii is derived from the foetus by transudation through its skin is also, I think, sufficiently disproved by the observation of Schroeder, already quoted, as to its presence in amniotic sacs where the foetus has become wholly blighted. If, therefore, the liquor amnii proper, the liquor amnii as it exists before receiving the contributions referred to, be not of foetal origin, it must of necessity be derived from the mother, and among the maternal structures which can produce it, it would appear unnecessary to look beyond the amnion itself. The amnion is a serous membrane both in its derivation and structure; its derivation being from the serous layer of the blastodermic membrane, and its structure a layer of pavement epithelium, resting on a fibrous basement membrane, attached by a layer of connective tissue to the subjacent chorion. It has, therefore, every requisite for the secretion of a serous fluid, its lining of epithelial cells, as suggested by Dr. Priestley, being the immediate agents in the process; and if this view of the source of the liquor amnii, sustained as it is both by the character of the fluid and by the character of the sac which contains it, be accepted, then its occasional excess should be as capable of explanation as in the case of serous effusions elsewhere.

The majority of cases, if not all, may, I think, be brought under one of three heads.

Under the first would come all those cases in which the excess is due to an inflammatory condition of the amnion. Many

such are reported; but as to their relative frequency much difference of opinion exists. McClintock, in his memoir on the subject, speaks of opacity and thickening of the amnion as exceptional. Schroeder, on the other hand, regards inflammatory proliferation of the membrane as usually the primary process. It would require the collation of a large number of observations to settle the point as to the exact proportion of cases of inflammatory effusion, but for our present object it is sufficient to note that many such are recorded. Caseaux indeed quotes from one author who gives particulars of cases in which not only was the amnion thickened, but with false membrane on its inner surface. This probably would be the cause in those cases of twin conception where but one sac is dropsical, and where, therefore, the cause must be localised in the amnion affected. These cases would be comparable with inflammatory effusions in other serous cavities, with ascites, for example, from peritonitis; or with hydrothorax from pleurisy. Under the second head would come those cases where the decidua has been found diseased and hypertrophied, but the amnion healthy, and the resemblance here would be to cases of effusion from obstructed circulation. If the decidua be so thickened, either as a result of inflammation or as an occasional sequence, according to Virchow, of constitutional syphilis, as to interfere with the passage through its tissues of venous blood, an effusion of serum will take place, which, by transudation through the amnion, will produce in its cavity an excess of fluid. It is in these cases that the nutrition of the foetus suffers most; that there is the greatest tendency to abortion, and that if gestation continue to term, the foetus is so commonly found atrophied or ill-developed; and then, lastly, hydramnios may most probably, like serous effusions elsewhere, be the result of some maternal blood dyscrasia. In this class would come, I think, many of those cases in which a tendency exists to recurrence of the dropsy in successive pregnancies. I have known two such cases in which the tendency persisted in two and three pregnancies respectively, and in which at an early period before kidney congestion could be the result of direct pressure albuminuria existed and continued for some weeks after the labour was over. In these cases there are no obvious pathological changes either in the amnion or the decidua.

Under one or other, then, of these classes it appears to me all cases of hydramnios may be brought; and, if so, the apparent antagonism of the different theories held as to the origin of the disease disappears, and is resolved into the fact that no one cause accounts for every case, but that in some it may be an inflammatory effusion, in others connected with disease of the membranes external to the amnion; and in others the result of a maternal toxæmia.

## KAKKE.

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THIS disease, as known in Japan, is almost entirely new to the European medical world, the only published description of it being a paper by Dr. Hoffman (late physician to the civil hospital and medical school of Yedo) in the July, 1873, number of the 'Transactions of the Deutsche Gesellschaft für Natur und Völkerkunde Ostasiens.'

Kakke is a disease of considerable interest, not only from the prominent part which it plays in the nosology of Japan, but also from its probable identity with the terrible affection known in India, Ceylon, and the south of Brazil as iseriberi.

The name "kakke," derived from the words "kiaku" and "ki," the Japanese pronunciation of two Chinese characters signifying "leg," and "condition" or "disease," indicates merely the seat of the most common symptoms of the ailment. The earliest account extant of kakke in Japanese literature is in the 'Zatsu biyo kibun,' a work published in Kiyoto in the year 1715, written by a physician named Tachibana Nan'ke; it is, however, probable that the disease had then already existed for a long time, but did not occur with sufficient frequency to cause it to be recognised as a separate affection.

Kakke may be defined as follows:—A recurrent, non-febrile, non-contagious disease, endemic in certain low-lying towns of Japan, and especially associated with over-crowding, bad drainage, and bad ventilation; most prevalent during the period

of high temperature and heavy rainfall, capable of remaining latent for very long periods, and of manifesting itself under ordinary exciting causes in places remote from its source. The symptoms are characterised by temporary numbness of certain portions of the surface ; paralytic affections of various muscles, most commonly those of the extremities ; the loss of power, sometimes associated with spasm, muscular hyperæsthesia, and progressive atrophy ; dropsical effusions, usually slight and limited to the subcutaneous connective tissue of the lower extremities, sometimes extensive, and involving serous cavities, especially the pericardium ; reflex vomiting in the most acute cases ; abnormal excitability of cardiac motor centres leading in ordinary cases to palpitation, in acute cases to extremely rapid action of the heart, and consequent exhaustion of the organ, failure of circulation, and death.

*The symptoms* are varied but characteristic, and enable us to arrange the mass of the cases under three chief headings, acute, subacute, and chronic, but it must be understood that no defined line can be drawn between these, as both the acute and chronic conditions commence with subacute symptoms, and many intermediate forms are met with.

In the present description the phenomena of the disease will be first enumerated, and afterwards an example of each of the three forms selected from the cases under treatment at the naval hospital of Yedo during the past year will be given in detail.

#### *Disorders of Sensation.*

*Cutaneous anæsthesia.*—This most commonly affects the lower extremities, abdomen, upper extremities, and lips. The lower extremities are almost invariably involved, but one or more of the other situations named are also implicated in a large number of instances ; in exceptional cases the sensation in other localities is impaired. It shows no tendency to favour any particular nervous area in the parts attacked ; thus, in some cases the condition is most marked on the inner side of the leg, in others on the outer, in others, again, it may be referred to the anterior or posterior aspects. In the upper extremity it is usually first manifested by clumsiness in buttoning or adjusting the clothes.

The degree of anaesthesia is very variable; it is sometimes so slight as to be difficult to measure accurately, at others there is almost complete insensibility. At the tip of the index finger the separate contact of the two points of the aesthesiometer may not be perceived till the points are five or six lines apart, and on the extensor side of the leg a separation of six inches or more is frequently necessary.

Its duration is variable. It rarely, however, either in amount or persistence, bears any relation to the severity of the other symptoms, and may disappear altogether long before the disease is cured.

Pains in and around the joints are sometimes complained of and may become chronic. These are possibly rheumatic.

*Muscular tenderness*, as a slight and transient symptom, is common, but unimportant; when, however, it becomes severe and accompanied by cramps it forebodes atrophy of the affected muscles. The usual seat is the leg, but the thighs and upper extremities may also suffer.

*Spinal tenderness* is rarely present, but occasionally may be noticed over the dorsal or lumbar region.

#### *Motorial Disorder.*

*Paralysis*.—Marked loss of power in the lower extremities is an almost constant symptom. In slight cases it may be indicated merely by a sensation of looseness of the joints and early supervention of fatigue on slight exertion; in more severe cases the legs will no longer support the weight of the body, but even in the worst examples complete paralysis, except as a result of muscular atrophy, is unknown; excitability to electrical stimuli is frequently diminished, but not lost.

The upper extremities are less often involved than the lower, but the degree of loss of power is sometimes very great, the grasp becoming so feeble as to be scarcely perceptible to a hand placed within that of the patient. The two arms are commonly affected in an unequal degree. The impairment does not appear to predominate in any particular muscles in the affected parts as a general rule, but sometimes a special flexor or extensor may be selected for more complete paralysis than the rest. The

muscles of the abdomen are occasionally involved, in somewhat rare instances the laryngeal muscles suffer, and still more exceptionally the muscles of the tongue and pharynx. The facial, cervical, and thoracic muscles, and the sphincters of the bladder and anus, are apparently exempt.

*Spasm or cramps.*—A complaint of spasm in the muscles of the calf, very rarely elsewhere, is a common symptom, but is seldom severe unless muscular tenderness be also present.

*Muscular atrophy.*—In most cases the enfeebled muscles are soft and flabby, but in the slighter examples they soon regain their fulness and firmness under treatment. In the more chronic forms of the disease, however, a steady wasting occurs, nearly always associated with marked tenderness and occasional cramps. Where treatment has been neglected or unsuccessful a condition closely resembling progressive muscular atrophy may supervene.

The wasting process occasionally selects a special group of muscles, as for example, the extensors of the toes, the interossei of the hands, &c., but usually it is general in the weakened limbs.

In two cases in the naval hospital hard tumours have been seen in the substance of the flabby gastrocnemii, the mass disappearing as the muscles regained strength in both instances. The abnormality is probably an interfascicular proliferation arising from functional disturbance of the nerves which govern the nutrition of the part.

Microscopical examination of the affected muscles in the early stages of the disease shows no alteration of aspect, but, as atrophy goes on, the transverse and longitudinal striae of the fibres become less clearly defined, the sarcous elements maintain for a time their relative positions, assuming, however, the appearance of linearly arranged granules; in the latest stages of wasting the fibres are shrunken, granular, or fibroid, and show no trace of the original structure.

*The intellect* is unaffected in *kakke*.

#### *Circulatory Disturbance.*

*Palpitation* is complained of in about two thirds of the cases; it is mostly slight and occasional, but sometimes, especially in

the acute form of the disease, forms a prominent symptom. It nearly always accompanies anaemia, but may exist where there is no evidence of a deficiency of red corpuscles. The attacks of palpitation originate independently of exertion, but are aggravated by it.

*Alteration of heart sounds.*—Murmurs usually systolic, most audible either at the base of the heart and in the great vessels, at the apex, or at the epigastrium, are common, and a diastolic murmur over the semilunar valves is not rare. Simple prolongation of the first sound and duplication of the second sound are also frequent. These abnormalities are all temporary and disappear completely under treatment in the cases which recover. They have no apparent relation to anaemia and may be absent when the patient has an almost bloodless aspect, and present when no appearance of anaemia can be detected.

In acute cases the heart's action becomes remarkably altered, and the whole circulation appears to be thrown into disorder. The cardiac beats are extremely rapid, irregular, and unequal, associated with visible and palpable thrill and loud diffused murmur in some cases, in others with signs of pericardial effusion. There is a sense of constriction at the lower part of the thorax, and a state of the most intense "anxiety." A visible pulsation in the neck is often associated with extreme feebleness and smallness of the radial and posterior tibial pulses; orthopnoea and cyanosis are present, but without marked venous distension, the circulation in the extremities becomes less and less perceptible, the hands and feet become cold to the touch, chilliness extends towards the trunk and death soon follows, the intellect remaining clear almost to the last.

*Sphygmographic tracings* are mostly negative, the only peculiarity noticeable, being, in a certain proportion of cases, low arterial tension, which disappears as the disease progresses towards recovery.

*Endocarditis and pericarditis* are said to occur occasionally, but post-mortem evidence of these is wanting at present. I have not yet met with any satisfactory evidence of either condition during life.

*Respiratory disturbance.*—This when present appears to be secondary to the circulatory disorder. The frequency of the

respirations is increased while palpitation is going on. The state of the respiratory functions during the most acute symptoms has just been described.

*Disorder of the digestive system.*—Signs of gastric irritation are very common, but dyspepsia in all forms is so general a condition among the Japanese, that the furred tongue with injected papillæ and the nausea, and epigastric tenderness found in about 5 per cent. of the cases of kakke cannot be considered as having any special bearing upon the disease. Vomiting from simple gastric disturbance may occur, and must be carefully distinguished from another kind of vomiting to be presently referred to.

*Loss of appetite.*—Constipation, &c., are frequently found, but are not constant. The hepatic functions present nothing for notice.

*Splenic enlargement* is very rare. It has been noticed in one case at the naval hospital, but in this the coexistence of a certain amount of fever rendered it probable that the kakke was complicated with ordinary malarious poisoning.

*The vomiting* of kakke as distinguished from common stomachal irritation is one of the most portentous symptoms of the disease. Sometimes it ushers-in the acute circulatory disorder previously referred to, at others it is followed by a rapidly fatal exhaustion altogether disproportionate to the violence or frequency of the act. In many cases it may cease entirely after the exhibition of remedies, but unfortunately its cessation is seldom followed by any improvement in the condition of the patient. It appears as a rule to be rather an indication of a grave alteration in the system than dangerous in itself, but sometimes it undoubtedly, by inducing exhaustion, takes an active share in the fatal termination of the disease. It is apparently central in origin and is usually unaccompanied by nausea or any sign of digestive disturbance, but acute epigastric pain and tenderness may be present.

The expelled matters consist of food if any has been recently taken, of a little bile and mucus; not unfrequently an *ascaris lumbricoides* (the most common of the intestinal worms of Japan) is found in the first ejecta.

*Dropsical effusions* play an important part in the symptomatology of the disease, but there is reason to believe that their

share in the production of a fatal result has been greatly exaggerated. The effusion most commonly shows itself in the form of oedema of the legs. This condition is one of the most constant signs of the disease ; it is frequently, however, very slight and of very short duration, and hence is often absent when the patient presents himself for examination, although the impairment of sensation, loss of power, &c., may be well marked. The indications of its presence are usually limited to a little pitting on pressure over the tibiae, most distinct over the posterior border of the subcutaneous surface ; it seldom extends to the feet or even to the malleoli, a peculiarity which appears to distinguish it from oedema due to other causes.

In a certain number of cases, however, the effusion becomes more widely distributed, involving the face, neck, chest, and even the whole subcutaneous areolar tissue, but rarely, if ever, reaching the degree common in renal anasarca ; well-marked anaemia is nearly always associated with this condition. The face, next to the legs, is the part most subject to oedema, which here shows itself mostly by a slight aspect of puffiness easily passed over without notice by an unpractised eye. The upper extremities are least frequently affected. The effusion is occasionally somewhat abruptly localised to a portion of the trunk, the front of the thorax especially, and in these cases, as noticed by Dr. Hoffman, the other symptoms are apt to be of a serious type.

Accumulations of fluid in the serous cavities occur in some of the worst cases. Hydropericardium, hydrothorax, or ascites may develop rapidly and be readily detected by physical signs, but in nearly the whole of the severe and fatal examples which have been in the naval hospital during the past two years the symptoms of intense circulatory and respiratory disturbance have been quite unconnected with dropsical effusion, and where fluid can be detected in the pericardium or pleura, it is probably related to the concomitant circulatory and respiratory phenomena as a coincident effect of a common cause—irritation of the sympathetic centres. It may, however, be sufficiently extensive to seriously aggravate the symptoms, and even to be the immediate cause of death. I am informed that the pericardium was tapped on two occasions by Dr. Hoffman, and a considerable quantity of fluid removed, but without interrup-

tion to the fatal course of the symptoms. *Œdema* of the lungs is more rare than effusion into serous cavities, but occasionally occurs in the course of the acute symptoms and is then rapidly fatal.

*The renal functions* are not especially affected in kakke. During the process of dropsical effusion the quantity of urine is diminished and the proportion of solids increase, while the reverse condition attends the reabsorption of the exudation. There is no albumen or diabetes.

*The cutaneous functions* present no points of importance for notice.

*The blood* has, I believe, never been analysed chemically, but microscopical examination shows no abnormality. When a patient has died from the acute symptoms previously described the blood is dark and feebly coagulable, but not otherwise peculiar.

The ordinary signs of anæmia, pallor of the lips and conjunctivæ, &c., are noted in our records as accompanying 10 per cent. of the cases ; the presence and degree of apparent anæmia, however, bear no obvious relation to the severity or duration of the kakke symptoms.

*Febrile disturbance* is so far exceptional that in the small proportion of instances in which a rise of temperature is noted we may fairly assume that it is due to some complication not forming a part of the disease under consideration. An elevation of  $2^{\circ}$ — $4^{\circ}$  Fahr. is occasionally seen, but its subsidence does not coincide with or herald any marked change in the other symptoms. When acute symptoms set in the temperature (taken as in the axilla) usually falls to  $1^{\circ}$  or  $2^{\circ}$  Fahr. or even more below the normal level.

The *duration* of the disease ranges from about two weeks to twelve months or even more. The average period of treatment in the common subacute cases is about five weeks.

*Death* occurs from exhaustion or from acute respiratory and circulatory disorder.

Exhaustion may be rapid, as in cases in which persistent vomiting forms a prominent feature, or very prolonged, in chronic cases where the motor paralytic symptoms and muscular atrophy steadily progress, and general nutrition becomes secondarily impaired.

The mingled asphyxial and asthenic symptoms of acute cases appear to originate in extreme functional disturbance of the heart, assisted in some cases probably by the direct mechanical effect of pressure of effused fluid. Where oedema of the lungs or extensive hydrothorax occurs the phenomena are more purely asphyxial, but in my experience a functional cardiac disorder plays an important part in every fatal case, and is usually the immediate cause of death.

The following reports are quoted as fairly typical examples of the three forms in which the disease is most commonly found. In the first the history and description given would suffice with little alteration for more than one half of the cases that come under notice.

**Case 1.**—Nakamura, *st. 28*, marine, admitted into the naval hospital on the 18th June, 1875, suffering from a third attack of kakke contracted at Yokohama.

The illness commenced five days before admission with numbness of the outer sides of the legs, and a little oedema over the tibiae, at the same time fatigue of the lower extremities and occasional spasm of the gastrocnemii were noticed after ordinary exertion ; during the next two or three days the numbness extended to the lips and tips of the fingers, palpitation set in, and the oedema, spasm, and loss of power of the lower extremities increased, the knee-joints felt loose during the erect position. A slight sensation of general debility was complained of.

On admission his condition was as follows :

General aspect that of a fairly healthy man, no perceptible anaemia. Heart's action frequent but regular ; there was, however, occasional troublesome palpitation. A systolic bruit of a diffused character was audible over the apex and less distinctly upwards towards the base. No signs of hypertrophy. Pulse 98, rather small and feeble. Inspiration normal. Digestion impaired, slight epigastric tenderness, tongue furred, papillæ at tip abnormally red and prominent ; appetite bad ; bowels constipated. Slight anaesthesia of outer side of both legs, of tips of the fingers, and of lips. Gastrocnemii flaccid, but not tender, movements of lower extremities free in all directions, but feeble ; the patient could now only totter a few steps. Slight.

pitting on pressure over the inner border of tibiae, not over malleoli or feet. Urine normal.

*Treatment.—*

B. Mist. Sennæ Co. 3ij, et  
 B. Tinc. Ferri Perchlor. m<sub>xv</sub>,  
 Liq. Strychniæ m<sub>v</sub>,  
 Aq. 5j.

To be taken three times daily after food.

Electricity (magneto-electrical machine) to be used twice weekly.

The patient was discharged cured August 23rd, 1875.

CASE 2.—Omura, æt. 27, sailor, admitted August 2nd, 1875, with a first attack of kakke. The patient was in good health until three weeks ago, when he noticed fatigue in the lower extremities and spasm of the gastrocnemii after walking a short distance, the legs became slightly œdematosus and a sensation of numbness appeared over the distribution of the external saphenous nerve, and afterwards over the abdomen and tips of the fingers. The power of the upper extremities then became impaired, palpitation set in, at first after, but eventually independently of exertion. The œdema and palpitation subsided gradually before admission, but the loss of power increased.

*On admission.—* The patient was rather feeble, but not perceptibly anæmic. Heart sounds normal, pulse soft and rather frequent. Appetite and digestion fairly good, tongue clean. Temperature normal.

The numbness in the lower extremities, abdomen, and tips of fingers still remained, but was very slight. Muscles of upper and lower extremities somewhat shrunken, especially the right gastrocnemius; all ordinary movements could be performed, but slowly and feebly; great tenderness on pressure over the weakened muscles; the right gastrocnemius is acutely sensitive to touch or pinching. The whole of the muscles of the upper extremity were markedly feeble, but could be used for ordinary purposes requiring little strength; they were slightly sensitive to pressure. Severe and constant pains with tenderness on firm pressure in the neighbourhood of the knees were complained of. The muscles contracted on application of electricity, but considerable spasm and pain followed its application. The cutaneous numbness disappeared a few days after admis-

sion, but the further progress of the case was very tedious. During the period of eight months the atrophy steadily went on, the muscular tenderness remained unabated, and pain and spasm were so severe as to require daily administration of morphia by injection ; the general debility somewhat increased, but in other respects the constitutional condition was unchanged. The remedies given during the period were useless.

On the 10th of April the exhibition of tincture of aconite in 15-minim doses, repeated three times daily, was commenced. Its effect appeared to be almost immediate, a steady diminution of the symptoms followed, and now (June 10th) the muscular tenderness has entirely disappeared, the muscles are recovering their fulness and tone, and the patient is able to walk with the assistance of a stick.

The characteristic of this case is the almost complete localisation of the symptoms in the muscles of the extremities, the numbness, œdema, and palpitation having disappeared in an early stage of the disease. The rapid effect of the tincture of aconite is worthy of notice. Similar, but less speedy, results have been since observed in many other examples of the chronic form of the affection. In many chronic cases more or less general œdema with anaemia and evidence of circulatory disturbance are associated with the paralytic symptoms for several weeks or even months.

**CASE 3.**—Yoshida, æt. 21, marine ; admitted November 18th, 1875, with a first attack of kakke of five weeks' duration.

The disease commenced with numbness of the legs, fingers, and abdomen, feebleness of both extremities, occasional spasms in the legs, and frequent palpitation. No œdema was noticed. General debility appeared during the course of these symptoms, but did not precede them. Five days before admission vomiting after food set in, but subsided after three days.

*Condition on admission.*—Aspect anaemic ; no emaciation. Pulse soft and feeble, heart sounds normal at time of examination, but severe palpitation was said to occur from time to time. Digestive system disordered, appetite bad, and eating was followed after a short interval by nausea ; tongue slightly furred, papillæ at tip very red and prominent ; slight pain and tenderness in epigastrum and right hypochondrium ; thirst.

Upper and lower extremities very feeble, power of grasp in

right hand weakened, in left almost lost. The legs would not support the weight of the body. Numbness of front, outer side, and back of legs, of front of thighs, of forearms, and of lower part of abdomen. No muscular tenderness.

The patient was treated at first with alkalies and bitter tonics until the symptoms of stomachal disturbance disappeared, afterwards by iron and strychnia, but without producing further improvement.

On the night of the 23rd of November vomiting suddenly occurred, and was repeated at short intervals for about four hours, when it ceased, apparently rather from muscular exhaustion than from the remedies (ice, kreasote, &c.) which were given. The ejecta consisted at first of food, afterwards of bile and mucus. The vomiting was unattended by nausea, the tongue was clean, and there was no epigastric tenderness. Severe constrictive pain around the lower part of the thorax, irregular and rapid action of the heart, dyspncea, restlessness, and intense "anxiety" appeared almost immediately after the commencement of the sickness, and continued unchanged after its subsidence. The bowels were opened at about 7 o'clock in the morning, and a little milk was taken and retained. At 9 a.m. the condition of the patient was as follows:—Face pale and dusky, lips dark, expression that of terrible distress, body incessantly changing its position as if in search of relief to some extreme suffering; abdomen tender on pressure; tongue rather dry; skin of face and chest warm, but extremities cold; heart's action rapid, feeble, and irregular, pulsation visible over whole cardiac area, and accompanied by a peculiar diffused vibration distinctly perceptible to both sight and touch; limits of dulness on percussion normal. Pulse thready at wrist and difficult to count, comparatively strong in carotids; visible pulsation in neck, veins not prominent. Temperature in axilla 97° (registration difficult on account of the patient's restlessness). Respiration laboured, irregular, short, and shallow, 48 per minute, interrupted by frequent sighing and inarticulate complaints. Pulmonic regions resonant on percussion; air entered lungs as freely as the character of the respiratory act would allow. Intelligence perfect.

Alleviative measures of various kinds were tried, but the cardiac and respiratory action became more and more enfeebled,

the radial and posterior tibial pulses became imperceptible, the coldness of extremities increased, at 11 o'clock the restlessness became replaced by apparent insensibility, and a few minutes afterwards the heart ceased to beat.

No post-mortem examination could be obtained. The points of interest in this case were the absence of dropsical effusions throughout the disease, and the sudden onset of the acute symptoms which commenced with reflex vomiting (the "kakke" vomiting may be compared with the previous vomiting from gastric irritation). The cardiac symptoms might, in the absence of careful physical examination, have been easily mistaken for those of the sudden pericardial effusion which forms a complication in a large proportion of the acute forms.

*Period of latency.*—The period during which the germs of the disease may remain in the system without giving any signs of their presence is undoubtedly very long, and has a range extending from a few days to several months. The following account of an outbreak under peculiar circumstances will serve to illustrate this.

In the beginning of November, 1875, a training ship, the Tsukubakan, left Yokohama for San Francisco with a crew of 250 men, 60 officers and cadets, and 3 English instructors, and returned on the 14th April, 1876. Throughout the voyage cases of kakke appeared at the rate of about two or three in a week, although the ship was distant from any possible source of infection, and was itself kept in a perfect state of cleanliness, and despite the more remarkable fact that the period of the year was precisely that in which kakke is most rare in Yedo. One fatal instance happened just before the arrival of the ship in Yokohama, upwards of six months after the patient had left Japan. The total number of cases was about sixty, all of which occurred amongst the crew, the officers and cadets, who were better lodged and fed, being quite exempt. The determining cause appeared to be the over-crowding which must always exist on a fully manned ship, and this was probably assisted by unwonted exposure to weather, and possibly by a diet which, though unobjectionable under ordinary circumstances, was not sufficiently nutritious to maintain the amount of vigour requisite to resist the depressing conditions of sea-life.

*Frequency.*—It is impossible to form any calculation as to the  
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prevalence of the disease amongst the civil population, but it is known to be very great; in the army and navy, however, fairly reliable statistics are attainable.

The reports of the military and naval hospitals of Yedo for 1875 show the number of admissions for kakke to be 660, or 3·8 per cent. of the total forces (about 17,500). Besides these cases, however, a very large number of men are invalidated as suffering from kakke, but from various causes (usually slightness of the symptoms) are not sent to the hospitals; if we include these the total proportion amounts to 26 per cent.

In the military stations of Osaka, Kumamoto, Hiroshima, and Nagoya, which are situated at the south of Japan, the average proportion of hospital admissions for kakke is 3·8 per cent., but the whole number reported sick from this cause forms no less than 33 per cent. of the total force of 15,000. In Hiroshima among 3445 men no less than 1844 cases of the disease appeared during the year. In Sendai, on the eastern coast, a similar ratio is reported.

*Mortality.*—The death rate can be ascertained only in the army and navy. Amongst the civil population it is probably much higher than in the public services, as the townspeople have none of the advantages of hospital accommodation and medical treatment which the soldiers and sailors possess.

The statistics of the army department show the number of deaths in 402 cases treated in the hospitals at Yedo during 1875 to be 89, or 22·13 per cent., and 25 men, or 6·25 per cent., were discharged from the service as "invalids." In the naval hospital, out of 286 cases treated during 1874 and 1875 the deaths were 15, or 5·15 per cent.; in the year 1875, however, the cases admitted were far more severe than in the previous year, and the mortality was 8·6 per cent.

The army returns for the whole of Japan show a mortality of 17·65 per cent. of the cases treated in hospitals. In Sendai and Kumamoto the rate was highest, about 30 per cent., in Nagoya lowest, 13 per cent. The death rate of beriberi in India ranges from 14 per cent. to 36 per cent., reaching the highest rate in the jails.

*Etiology.*—The causes of kakke, like those of beriberi, have been the subject of much speculation which has led to little definite result.

Beriberi is most commonly believed to depend merely upon a peculiar form of anaemia and debility induced in tropical climates, but this theory will not explain the occurrence of kakke in Japan, as the disease is known in Hakodate, which is nearly on the same isothermal line as London, and the climate of Yedo, the chief seat of kakke, cannot be considered tropical, as its mean annual temperature is not greater than that of Rome. Again, anaemia and debility, although frequent concomitants, are not at all general.

Most of the native doctors believe that the complaint is caused by some poisonous emanation from the soil, and hence, they say, the appearance of the earliest symptoms in the lower extremities which are nearer to the source of the evil influence than the rest of the body. Setting aside this argument in support of their theory, the view must be considered not unreasonable, as a striking analogy to ordinary malaria is seen in the relation between the prevalence of kakke and certain conditions of locality, climate, and season, and in the predisposition to repeated recurrence induced by the first attack; but the symptoms are totally different from those attributed to paludal poisons, and are not amenable to the same treatment.

The circumstances attending the development of kakke lead us to assume the existence of an atmospheric poison, but no single cause is sufficient to account for the generation of this; the essential conditions which combine to produce it appear to be overcrowding, neglect of hygiene, low damp localities, and a season in which high temperature is associated with heavy rainfall. When the germs have entered the system they are capable, as previously shown, of lying dormant for a variable period, their activity being commonly awakened by trifling causes which in a normal state of the system would cause little or no disturbance of health.

The various circumstances which appear to influence the development of kakke will now be considered separately, in order that the relative importance of each may be seen.

a. *Locality.*—Kakke is chiefly endemic in the three great cities of Japan, Kiyoto, Osaka, and Yedo, and was, until recently, supposed to be limited to these, but at the present time cases are known to occur at Hakodate and Nagasaki, the former being a Japanese settlement, a peninsula of the south of

Yezzo, and, hence, close to the extreme north of the main island (usually called "Nipon" in the European maps), the latter at the northern extremity of the island of Kinshin, and corresponding closely to the extreme south of the main island. It has also appeared amongst the troops stationed at Kumamoto, Nagoya, and Hiroshima on the south coast, and at Sendai over the east coast, and is common in Yokohama, the main European settlement within eighteen miles of Yedo. Further investigation is greatly needed, but there is little doubt that it would be rewarded by the discovery of the disease in a large number of other places; at present it seems to be peculiar to low-lying towns mostly on the sea coast, and having a damp, badly-drained alluvial soil. The vicinity of rice fields is usual, but not essential. In Yedo nearly the whole of the cases originate amongst the residents of the low, damp quarters of the city, while the disease rarely attacks the inhabitants of the more elevated parts. A moderate elevation (40—50 feet), however, if in the immediate neighbourhood of and especially if surrounded by low ground does not give complete immunity. It must be noted that the lowest parts of the city are also the most crowded, and are peopled, as a rule, by the poorer classes. A difficulty exists in the estimation of the effects of locality in the fact that the disease may make its appearance in places remote from that in which it has been contracted, and hence people of Yedo, Osaka, Kiyoto, settling for a longer or shorter time in other towns, may import it into these places. The native population of Hakodate and Yokohama is chiefly composed of emigrants from all parts.

The natives of places in which kakke is unknown become liable to the disease on settling in a district in which it is endemic, but not until the expiration of a certain period. The length of this period varies greatly according to the circumstances in which the new comer is placed, and to his powers of resistance against unhealthy influences; it is seldom less than six months, and averages about a year; in one case, however, occurring in the navy, that of a sailor who had previously lived in a healthy place, the disease appeared after only two months' residence in Yedo. It is remarkable that kakke does not occur in China, in which the conditions for its development appear to exist in a marked degree.

*β. Season and climate.*—During the dry healthy months from October to May very few cases of kakke occur, and there are probably instances in which the germs have been long dormant, but during the prevalence of the south-west monsoon, when high temperature is associated with heavy rainfall, the disease rate rises, the early cases appear at the beginning of June. Occasionally one or two weeks before this in July the number is largely increased, and in August the maximum is reached, a sensible decline is noticed towards the end of September. In the months of December, January, February, and March an outbreak is very rare.

The rainfall in Japan, roughly calculated, is about three times as great as in England, and usually occurs to the largest extent in July and August, when the temperature reaches its highest point; in these two months about one half of the total yearly number of kakke cases come under treatment. A fairly definite ratio between the height of the thermometer and the development of kakke may be constantly observed; it is especially worthy of notice that during June and July of the present year, in which the temperature has been unusually low as compared with the ten previous years (the rainfall *presenting* no peculiarity), the prevalence of kakke has been remarkably reduced; in the naval hospital the number of admissions being less than one third of the numbers for the corresponding months of 1874 and 1875.

Under certain circumstances kakke may make its appearance in the winter months and in cold latitudes, but only in persons who have been previously residing in kakke districts. An example of this is shown in the case of the crew of the *Tsukubakan* already narrated.

*γ. Hygiene.*—Bad drainage is a conspicuous evil of nearly every Japanese town, and *especially* in the low densely populated parts. Refuse matter is conveyed away by means of open gutters which run along the narrow streets close to the houses; these frequently become blocked and give off in the hot weather offensive and injurious effluvia, while the putrefying fluids arrested in their course percolate into the earth beneath the raised ground flooring of the wooden houses, and may reach the walls and mingle with the drinking water.

The principles of ventilation are entirely ignored in the

ordinary Japanese house ; during the day the rooms are fairly open, but at night the whole building is converted into a kind of closed box by means of wooden shutters, and fresh air can only enter through imperfections of structure.

Overcrowding is undoubtedly the most powerful of all the determining causes of kakke. In India and Ceylon beriberi makes terrible havoc in the barracks and jails, and in Japan kakke appears to reach its highest degree of prevalence and malignancy under similar circumstances. It is only to overcrowding in ill-constructed barracks that we can attribute the fact that large numbers of men, young, and chosen as free from disease and feebleness of constitution for the service of the Imperial Army and Navy, suffer even more than the weakly and often ill-fed members of the lower trading class. The defective sleeping accommodation of ships appears to be especially active in causing an outbreak, as in the case of the Tsukubakan. Another striking example occurred in the summer of 1875, in which an epidemic of kakke and other diseases attacked the crew of a Japanese vessel lying at anchor near Yokohama. Out of 300 seamen 97 were incapacitated for duty at one time, 47 of these suffered from kakke, and before any inquiry was instituted over 20 had died from the same complaint, the remaining 50 were prostrated chiefly by disorders of the alimentary canal. The food, clothing, and exercise of the men was found to be sufficient, but at night the whole crew, except a few kept upon deck, slept at one time in a space allowing only 32 cubic feet per head, while the anchorage of the ship and hilly conformation of the shore prevented anything like free renewal of the contaminated air. After a medical investigation the sleeping arrangements were entirely altered by the Admiralty authorities, and as a result the epidemic almost immediately subsided.

*Race.*—The disease has up to the present time been limited to the Japanese, no well-authenticated instance of its occurrence in the foreign community having been recorded. The safety of Europeans and Americans is probably owing to the absence, in their case, of the unhealthy conditions under which the complaint arises in the natives. The foreign troops stationed in Yokohama occupied well-constructed barracks situated on high ground. Almost all the civil residents are in good cir-

cumstances, and none are subjected to the overcrowding so common amongst the Japanese. In India and Ceylon the foreign troops have suffered extensively from beriberi, in consequence probably of defective barrack accommodation, and there is little doubt that outsiders placed under unfavorable circumstances in Japan would develope kakke.

*Age.*—An attack rarely occurs before the age of fifteen, and is comparatively uncommon after the age of forty. The period in which the greatest liability exists is between twenty and thirty.

*Sex.*—Women are less subject to the disease than men; the proportion is said to be about 1 to 3. It most commonly arises during the three weeks' confinement to bed, which is "de rigueur" for every Japanese mother after parturition.

The greater frequency of kakke in men probably depends upon overcrowding in barracks and ships in the case of soldiers and sailors, and upon the lazy sedentary life led by a large proportion of the males of the trading class, while the wives are saved from inaction by the necessity of carrying on the household duties and ministering to the wants of their husbands and families.

*Rank and occupation.*—The higher classes of the Japanese seldom suffer from kakke, probably because, in addition to the ordinary advantages of superior circumstances, they possess residences in the higher and more healthy parts of the town. Exceptions to the rule, however, exist as in the case of the thogun (Tycoon) Tyemochi, who is said to have died from the disease at Osaka eleven years ago, but on this as in many other instances it is possible that the diagnosis was incorrect, as formerly every affection in which dropsy or cardiac disturbance played a part was called kakke by the Japanese physicians.

The lowest classes, the coolies, are also remarkably exempt, but these people, owing to their laborious occupations in the open air, are the best developed and most vigorous men in the country, and are unusually free from disease in general.

The subjects specially selected are soldiers, sailors, small traders, and their assistants, and the poorer class of literary men and students. Sedentary occupations appear to be injurious chiefly in involving confinement to unhealthy houses.

*Food.*—As beriberi exists only in countries where rice is

largely grown, the disease is sometimes attributed to the non-nitrogenous diet of the people. The same view is held by many with regard to kakke, but is without foundation, since the coolie class who live more exclusively upon rice than the soldiers, sailors, or trading classes maintain a high degree of physical development and are little liable to the complaint. The Chinese moreover, who are rice eaters, do not suffer; and the occurrence of beriberi amongst foreign troops shows that nitrogenous diet affords no protection from that affection. The coincidence of rice cultivation and kakke or beriberi probably indicates merely that abundant moisture is necessary for the production both of the grain and the disease.

Insufficiency of food may act indirectly by lessening the power of resistance to morbid influences, and the injurious habit, almost universal in the rice-eating parts of Japan, of swallowing the grains without mastication is a potent cause of dyspepsia and all its resulting evils.

The drinking water of Yedo has been analysed repeatedly, but without special result. The organic matter has been found in small proportion, and the inorganic salts are not excessive. It will be necessary, however, to apply the tests to water taken from the lowest districts and in the kakke season before its absolute harmlessness can be safely assumed; the remarks previously made on the drainage will show that the probability of contamination of wells is very great.

Other circumstances besides those already named have some connection with the appearance of the disease, although they have no bearing of importance upon its genesis. Any condition capable of inducing temporary constitutional disturbance may at times determine a manifestation of the effects of the previously latent poison. Exposure to cold and wet is one of the most frequent of these. Many of the patients give an account of having "caught cold" as the starting-point of their symptoms. Such exposure is probably the cause of the brief elevation of temperature occasionally seen at the commencement of the disease. A change of place, even from low to high ground or from an unhealthy to a healthy situation, has been known to be followed by an attack, the sudden alteration of atmosphere, temperature, and perhaps of habits producing a slight shock to the system, and the consequent explosion of the

hitherto inert materies morbi. The same phenomenon has been observed in ordinary malarious affections.

Debility from past illness sometimes appears to arouse the symptoms, but a complication here exists in the confinement to the house necessitated by the previous complaint. In the naval hospital, which is on high ground and well ventilated, the convalescence of acute disease is very rarely interrupted by kakke. "Puerperal" kakke is probably rather a result of impure air during confinement to the sleeping room than to the debility left by the labour.

*Pathology.*—Direct pathological investigation has hitherto been too imperfect to throw any light upon the nature of kakke. The obstacles to a full autopsy are unfortunately very great on account of the deeply-rooted prejudices of the Japanese against any interference with the bodies of dead friends or relatives.

In the few post-mortem examinations which have been made no abnormalities beyond dropsical effusions have been detected, but it is to be regretted that in no case have the nervous centres been fully investigated. The localities to which the symptoms would direct our attention as primarily attacked by the kakke poison are in the spinal cord, medulla oblongata, and sympathetic centres. It is possible, however, that in many of the situations in which there is indication of disturbance of function the disease would not be productive of changes sufficiently coarse to be made out by our present means of investigation.

The explanation of the most persistent symptoms, the motor paralysis of the extremities, must be looked for in the anterior vesicular columns of the cord, and in the chronic cases of kakke organic change visible by aid of the microscope would probably be detected. The spasms most noted in the early stages of the disease would have their origin in an irritation of the same parts.

A localised and usually transient affection of the posterior cornua would account for the numbness so constant on the commencement of the symptoms. The muscular hyperæsthesia is less easy to explain, as it may originate either in the cord or in the affected muscles themselves, most probably the latter, as it goes on *pari passu* with the structural changes in the muscular fibre during the atrophic process. The occasional

loss of voice without interference with the respiratory function of the larynx, interpreted by Bernard's experiments, would be a result of the paralysis of the internal or communicating branch of the spinal accessory. A still more rare manifestation, impairment of articulation, may be due to changes in the nucleus of the hypoglossal.

The dropsical effusions, nearly always peculiarly localised, and capricious as to situation, of course differ entirely in pathology from the renal and other ordinary dropsies seen in Europe. A more or less extensive implication of vaso-motor centres may be their probable explanation.

The cardiac disorder is probably referable to irritation of the origin of the cardiac accelerator nerve, leading in the worst cases to rapid exhaustion of the heart by overwork and loss of the nutritive pause. The vomiting of kakke appears to depend upon irritation of that portion of the sympathetic to which, according to the view of Longet, the motor action of the vagus upon the stomach is due.

It is hoped that opportunities of gaining more positive knowledge will soon be attainable; in the meantime an apology is offered for the introduction of suggestions in the place of practical investigations.

*Prophylaxis.*—The means of prevention of kakke, or, at least, the reduction of the extent of its ravages, is a question of national importance in Japan, as every year the lives of many thousands of the inhabitants of its chief cities are endangered, and the force of its army and navy is reduced to a very appreciable degree. The ill-effects are likely to be most seriously felt in the navy, as the disease tends to break out with most violence amongst men in active service, and would hence be liable to cause the largest amount of invaliding when the duties of the sufferers are of the greatest importance.

It has been already shown that a number of circumstances combine in producing the development of kakke; all of these we cannot hope to remove, and consequently we can have little expectation of an entire annihilation of the affection, but much may be done to reduce the number and severity of the cases. The means adopted for this purpose would also lessen the prevalence of many other diseases usually found in association with kakke, as enteric fever, &c., and would also elevate the

rather low standard of health and vigour in the population generally.

The methods which have been suggested for the improvement of dwelling houses, barracks, &c., of street drainage, and the hints, by no means unnecessary, offered in connexion with individual hygiene need not be recapitulated here. The attention of the Japanese Government is now being drawn to the subject, and it is probable that decided steps will soon be taken.

*Treatment.*—The curative treatment of kakke, according to rational therapeutics, has been fairly successful, the symptoms and sequelæ in the larger proportion of the more severe cases can be relieved, and the supervention of acute cardiac symptoms, so liable to take place in the course of seemingly mild attacks, appears to be frequently averted while the patient is under careful observation. The large experience of Japanese physicians has not as yet been productive of any original means of cure more efficacious than moxas applied to the oedematous parts; the administration of various drugs, the individual action of each of which is entirely unknown, and a somewhat modern theory of starving out the poison by means of low diet, but the newly educated men are now beginning to approach the disease with better weapons, and we may soon hope for rapid addition to our powers of conquering even the worst manifestations. The indications to be followed are, firstly, to remove as far as we are able the immediate and remote causes; secondly, to neutralise or eliminate the blood poison if possible; thirdly, to relieve special symptoms; and lastly, to maintain or improve the general health.

#### *1. Removal of the immediate and remote causes.*

The removable conditions mostly coexisting to determine an outbreak of kakke are overcrowding in badly constructed dwellings in low unhealthy situations; even in those cases where the affection appears in a place remote from that in which the poison was generated the immediate cause is almost invariably overcrowding combined with bad ventilation. Many of the Japanese medical men fully recognise the importance of change of locality as an element of treatment, and recent experience

abundantly proves that the conveyance of the patient to a well-built hospital on high ground will often supersede the necessity for drugs, and always is a most valuable contributor towards the cure.

### 2. *Neutralisation or elimination of the poison.*

Neutralisation of the kakke poison has been attempted extensively but unsuccessfully. Quinine has undergone the longest trial, but except as an ordinary tonic, or as a temperator in the few cases in which fever coexists, has been found useless. Arsenic is spoken highly of by Dr. de Silva Lima, of South Brazil, in beriberi, but statistics which show a mortality of 26 per cent. do not inspire confidence as to the efficacy of the drug as a specific remedy ('Gaz. Med. de Paris,' 1872). It has up to the present time been found inert in kakke. Sulphites, hypochlorites, carbolic acid, &c., have also given negative results. Treeak Farook, a remedy used in India, is said to be of service in the less severe cases of beriberi, but as such cases yield readily to other means, the introduction of the medicine into Japan is scarcely necessary.

The elimination of the poison by diaphoretics, diuretics, and purgatives has been tried repeatedly, but without other effect than that of weakening the patient.

### 3. *The relief of special symptoms.*

*Motor paralysis.*—*Strychnia* combined with the *perchloride of iron* is now used largely in the navy, and, as far as can be judged by general comparison with the previous results of other remedies, with very good effect. The disappearance of the muscular weakness is hastened and the other symptoms usually rapidly subside. Where muscular hyperesthesia and severe spasm, however, are present strychnia is injurious and may greatly aggravate the symptoms.

*Electricity* is used in the same cases as strychnia. The intermittent current has been found more successful than the continuous current. The most marked results are in chronic cases when advanced muscular atrophy remains but the spasms

and hyperæsthesia have departed ; the restoration of the wasted muscles to serviceable bulk and power is sometimes remarkably rapid. . The application is usually continued for about fifteen minutes, and repeated every day or every other day according to circumstances.

*Cutaneous anaesthesia* appears to require no special treatment ; it always subsides during the cause of the paralytic symptoms.

*Muscular hyperæsthesia*.—This with its almost invariable associates, spasm and atrophy, is one of the most obstinate of all the symptoms, and yields slowly even under the most favorable circumstances.

The remedy found most valuable is tincture of aconite in  $\frac{m}{xv}$  doses three times daily. This has in some instances produced an immediate and rapidly advancing improvement after other means had been tried fruitlessly for many months.

Iodide and bromide of potassium, the former in 10, the latter in 15—20-grain doses, have been of service in a few cases, but no reliance can be placed on their action.

Conium, belladonna, and ergot have been tried, but without success. Morphia injection as an alleviative is useful. Local applications to the spine, blisters, ice, &c., have not given good results.

*Circulatory disturbance*.—Palpitation in its milder form usually subsides without treatment ; when troublesome, digitalis generally gives relief. Morphia injection is a certain alleviative, but its effects do not last about three or four hours.

The *cardiac acceleration* of acute kakke often runs its course so rapidly and surely that the arrival of death seems to mock our feeble efforts to intercept him. Stimulants and antispasmodics have been repeatedly given by both Japanese and European physicians, but those who have had most experience of this treatment will, perhaps, pronounce it useless. The most hopeful drug theoretically is digitalis, but practically it appears to exercise no influence whatever. The subcutaneous injection of an alcoholic solution of the alkaloid would ensure the entrance of the remedy into the circulation, but this has not yet been tried. Venesection (to the extent of  $\frac{3}{x}$ ) has been practised in one case only, but no change in the symptoms followed.

*Digestive disorders.*—Gastric irritation of the ordinary type is relieved by bismuth and alkalies. Vomiting is more amenable to kreasote than any other remedy.

The *reflex vomiting* of acute kakke does not yield to stomachic remedies; its cessation from exhaustion is, however, often mistaken for an effect of drugs previously given. In some cases in which the reflex vomiting has been complicated with and aggravated by ordinary gastric irritation, kreasote has been found serviceable. In a few instances subcutaneous injection of morphia has been of use.

*Dropsical effusions.*—The slight oedema of the lower extremities seen in the milder cases commonly disappears before the patient comes under medical treatment. General oedema disappears slowly during the administration of diuretics. Scoparium and juniper have been found most useful for this purpose in the naval hospital.

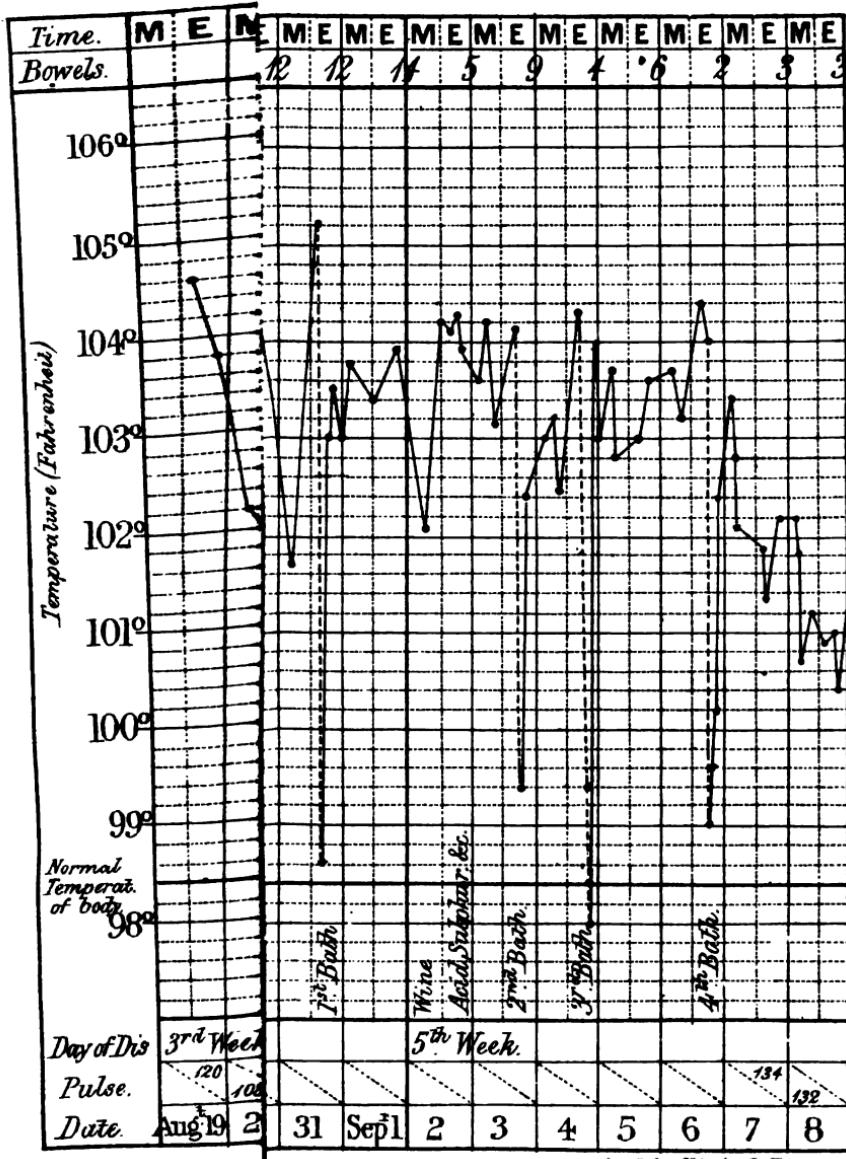
In hydropericardium, paracentesis has, as far as can be ascertained, been practised only three times, but in these cases the withdrawal of the fluid produced no relief to the cardiac symptoms. In hydrothorax and ascites the effusion is rarely sufficient to call for operative interference.

#### 4. *Improvement of general health.*

This is effected by the ordinary measures, tonics, nutritive diet, and pure air. In the larger portion of the cases, however, there are no general constitutional symptoms requiring medicinal treatment, but a liberal allowance of food assists the relief of the local manifestations.

# ENTERIC FEVER.

1. C (Charity Ward, No. 26.)

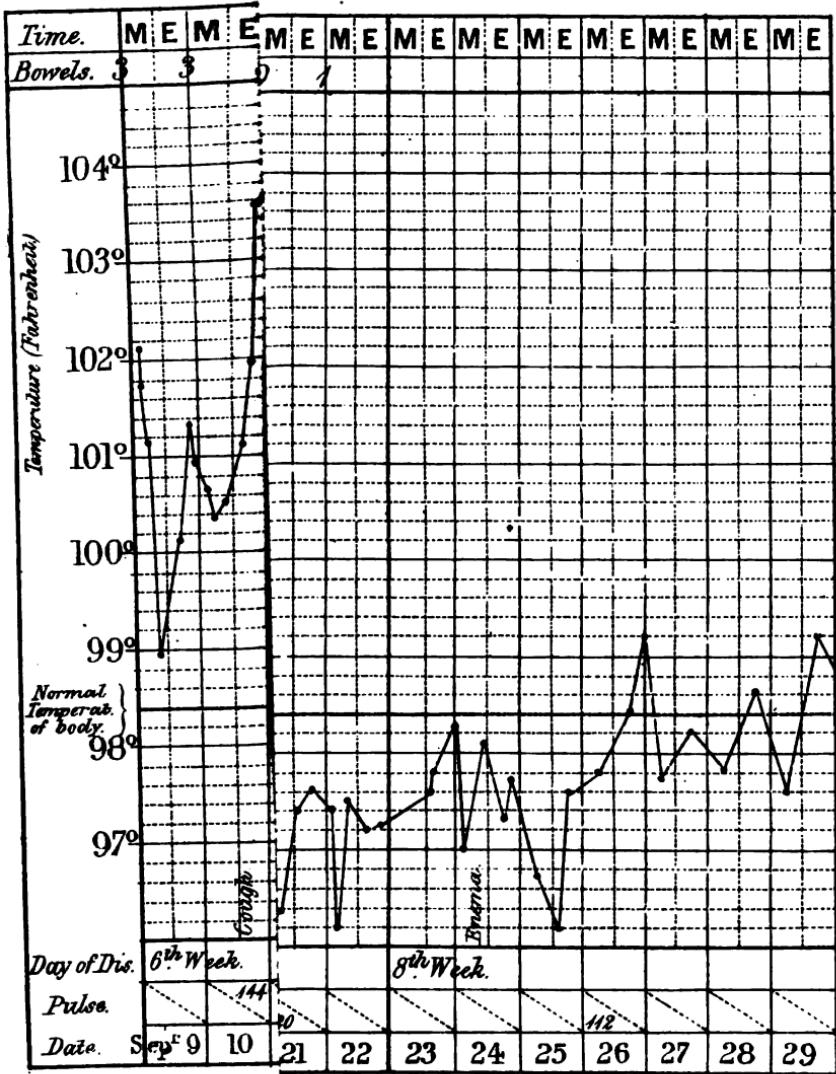


Gould's Clinical Chart.



2.

Case nued.



Gould's Critical Chart



## NOTES

ON

### CASES OF NERVOUS DISORDER.

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By W. M. ORD, M.B., F.R.C.P.

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1. *On some cases of reflex irritation of the skin, and on the part played by reflex irritation in producing certain morbid conditions of the skin.*

HERPES ZOSTER is cited by Dr. Brown-Séquard<sup>1</sup> as an example of a reflex change of nutrition set going by neuralgia. The association of severe local pain with the herpes has naturally been noticed by a long series of observers, for the clinical experience of all physicians must teach them to look for the herpes in all cases of severe intercostal pain. But the meaning of the association has not been certainly seen, as far as I know, until Dr. Brown-Séquard has pointed it out. To say with one author (M. Délioux, 'Gaz. Méd. de Paris,' 1855, Nos. 32, 33, 35, 39) that herpes zoster is a catarrhal neuralgia which has for crisis a herpes, or with another that the herpes is a secondary phenomenon dependent upon a rheumatic or dyspeptic neuralgia, is still only to put the association before the mind without proceeding to explanation. M. Parrot,<sup>2</sup> indeed, takes great pains to prove that the distribution of the pain and of the eruption correspond

<sup>1</sup> 'Lectures on Physiology and Pathology of the Central Nervous System,' p. 167. 1860.

<sup>2</sup> 'Considérations sur le Zona.' Paris, 1857.

more or less accurately with the distribution of certain cutaneous nerves. A key to the comprehension of the whole phenomenon is found when the local morbid condition of skin is recognised as due to an irritation of centripetal nerves. Complete comprehension, however, is even then not attained, for there remain to be discovered the nature and mode of the irritation of the centripetal nerves, the exact modification of local nutrition which is expressed in a herpes, and the meaning of the want of correspondence between the intensity and duration of the pain, and the intensity and duration of the cutaneous morbid process.<sup>1</sup>

I have had recently under care as an out-patient a man who illustrates three interesting points in the clinical history of herpes zoster.

He is a large-headed, pale, anxious-looking man of fifty-nine years, who three years ago was treated in the hospital for left hemiplegia, following an apoplectic seizure, and probably dependent upon cerebral haemorrhage. The hemiplegia gradually diminished till, at the end of a year, he could walk though with relative feebleness of the left leg; the arm had regained less power, and the face was still slightly paralysed. There was no affection of sensation. The speech and manner were indicative of weakened mental faculties and the head drooped swingingly to the left, like the too heavy head of a hydrocephalic child.

While in this condition he was attacked with herpes zoster. Two large patches with corresponding areas of intense burning pain were developed on the left side of the chest, one just over and below the lower angle of the scapula, the other midway between spinal column and sternum over the sixth, seventh, and eighth ribs. The first was compact, of the size of the palm of the hand, and after a very severe process of suppuration left a rather firm, white, raised, and pitted scar. The other was less compact, less painful, and left numerous depressions in the skin, unaccompanied by any hardening.

The above-described scar was and remains to this day, nearly two years after, acutely hyperaesthetic. The touch of the feather of a pen, a light brush with the tip of the fingers, and the contact of clothing, all produce an intolerable smarting

<sup>1</sup> See Mr. J. Hutchinson, 'London Hospital Reports,' 1866, and Barenprung, quoted in 'Brit. and For. Med.-Chir. Review' of January, 1862.

pain which not only makes examination very distressing, but deprives him largely of rest. The other scars are not hyperæsthetic; but over both areas the deep burning pain persists as at first.

The three points to which attention has been directed by the case are, 1, the influence of age and depressing agencies on the intensity and duration of pain; 2, the proportion between the pain and the eruption; 3, the occurrence of persistent hyperæsthesia.

It is well known that age, which tends to make all neuralgias more obstinate, has a very marked effect in adding to the duration of the pain of herpes zoster.

In patients over 50 one may generally expect the pain to survive the eruption for some length of time; and in still older patients I have seen the pain persisting at the end of two years in so acute a form as to simulate a pleurisy. Generally speaking, this persistence of pain bears proportion to the degeneration or enfeeblement of the individual. The individual under notice has marked arterial degeneration, of which his hemiplegia is a distinct consequence; his intellectual powers are manifestly degenerate; and finally herpes has chosen the paralysed half of the body for its seat. That the duration of the excessive pain should be also excessive is an illustration of the general principle, and favours the hypothesis of a central mischief, of a wasting or depravation of some ganglionic structure.

Secondly, the eruption, as far as I have seen it, is usually more extensive and more virulent in the young. The deepest ulceration that I have ever observed was in a healthy girl of 22, who suffered only short and insignificant pain. In the present case, however, the ulceration was wide-spread and so deep as to destroy in one area the whole depth of the skin, leaving much such a scar as would be left by a burn. The third point, of persistent hyperæsthesia, marks also a great intensity both of local and central irritability. To this must be added the fact that there is tenderness, but of a different character, between this area and the spine, at the point where the seventh and eighth intercostal nerves would send out their first cutaneous branches. The sufferings of this patient are certainly increased by cold and by excess of heat. He com-

plained bitterly in the hot weather of last August, as well as in the cold spring.

If we assume, as there seems fair ground for assuming, that the neuralgia is the cause of the herpes, by the agency of reflex nervous action, we have yet to explain the inversion of the ratio between pain and herpes in age as compared with youth; the general want of correspondence between intensity of pain, and extent and violence of local morbid action; and the fact that, after the herpes has disappeared and the ulcers left by it have healed, the pain persists without giving rise to more herpes. For relapses are certainly rare, and even if they occur the intervals are wide. There is almost suggested the occurrence of some change in the constitution of the local nerves, like a neuroma following an amputation—occasioned by the local process and giving rise to much of the pain and to much of the hyperæsthesia. It is certainly a fact that the best results in the treatment of this case—though palliation was all that could be attained—were due to the combined use of medicines and applications directed to the strengthening of the system, and soothing of the painful parts.

2. Eczema is known to be produced in certain cases by reflex action. Whatever may be the importance of the great factor of local hyperæmia, and whatever may be the power of local remedies in curing chronic eczema, the physiological origin of the local hyperæmia must be carefully studied if treatment is to be thorough and appropriate. To take an illustration from another affection of the skin—urticaria is clearly producible by certain external agents on the one hand and by certain medicines taken internally on the other, so clearly that probably no extreme holder of the opinion that skin diseases are chiefly to be treated locally would treat an urticaria dependent upon free stinging by nettles in the same way as an urticaria produced by poisonous shell-fish or by copaiba.

In citing these two latter instances I select illustrations of influences which appear to me to act on the skin through reflex nervous channels; and with urticaria produced reflexly by a substance which irritates the urinary organs and passages I wish now to compare eczema produced by irritation, mechanical and catarrhal, of the same system. I have notes of four cases

of eczema, one acute and three chronic, in which there seems to me clear evidence that the eczema was produced reflexly by internal irritation of this kind.

In the acute case the eczema broke out suddenly in a patient who had been for a long time the subject of vesical catarrh. He was attacked one day with pain in the region of the left kidney followed by symptoms indicating the passage of renal calculus. The diagnosis was verified by the appearance of several small oxalate-of-lime calculi in the urine after two days. But meanwhile a sharp burning irritation arose in the skin of the left side of the abdomen, chiefly over the track of the ureter, and shortly afterwards acute eczema appeared. Great fever followed and the eczema spread to the rest of the body, the part originally affected remaining the centre of the bitterest suffering. After the passing of the calculi there remained for some time considerable excess of vesical irritation, and not until this was subdued did the eczema yield.

A commentary on the reflex relation of the eczema is afforded by the fact that, in a subsequent passage of calculi attended with less renal and more prostatic pain, orchitis of the same side occurred. The eczema has also recurred twice, on both occasions in attendance on irritation of the urinary passages and in parallel progress therewith. The same patient has also had severe facial eczema following the application of a leech to the gums. In his case there is evidently great susceptibility in the reflex mechanism. He suffers severe pain in the lower limbs for long periods together, and has shown occasionally signs of the approach of reflex paraplegia. It is evident that there is general over-sensitiveness of the central nervous system, making it respond with unusual readiness to local irritations. The eczema, besides following in its development and decline one marked local irritation, is so remarkably grouped with the other instances of reflex irritation that it appears to me certainly of the origin here suggested. There are no indications of the operation of any diffused morbid poison in the case.

In the three other cases the suffering was less severe.

In one there is associated with chronic vesical catarrh, causing a permanently ammoniacal and muco-purulent condition of the urine, a prurigo of the front of both thighs which occasionally breaks out into a sharp neuralgic pain. When the

pain occurs, what the patient calls "shingles" follow. The so-called shingles are really an eczema with somewhat large vesicles; and the outbreaks are always associated with increase of the vesical symptoms.

In a third case eczema, chiefly of the legs, is connected with a pretty constant calculous irritation of the right kidney, and follows its variations. In a fourth, eczema complicated with boils appeared and disappeared in evident march with vesical catarrh.

In all these cases the eczema was best held in check, and in the last removed by remedy of the urinary trouble. In the second and third cases the urinary trouble was not curable by any treatment used, and though the recognised topical treatment of the eczema was used nothing more than temporary amendment was procured.

In several other cases I believe the same chain of causation of eczema to have existed; but only the above-cited cases were free from disturbing complication. In women there is clearly a reflex relation between the state of the mucous membrane of the genital passages and the skin of the body. In younger women there occurs frequently a form of acne which is almost pathognomonic of catarrhal state of the cervix uteri and upper part of the vagina; and most probably the pimply condition of the skin of the face, back, or nates which frequently appears in both sexes at puberty, resists all treatment and disappears spontaneously usually long after treatment has been discontinued is in reflex sympathy with activities in the genital passages incidental to the period of life. Similarly the psoriasis and the prurigo which are common at or about the climacteric period in women appear to me reflexly related with the local changes of that period, with the irritations of abortive effort in the one case, and with the failing local vigour in the other.

I pass over the reflex relations of eczema with gastro-intestinal conditions, and come to its fellowship with bronchial catarrh. This as far as I know is chiefly an alternative sort of partnership. There are now under my notice in our out-patient department two women in whom chronic eczema of the legs alternates with bronchial catarrh. In one of them whom I have seen often during the last two years the eczema is an affection belonging to warm weather, the catarrh to cold. This

is of course what might have been expected, but what is equally noticeable is the tendency of the bronchitis to appear independently of the weather when the eczema is checked by treatment. The two catarrhal inflammations appear to be related with a common cause or with common causes, probably both uterine and digestive, and each is capable of being by itself a measure of the distant excitant. The vaso-motor disturbance being hindered in the one district is transferred to the others ; that is to say, there being a tendency to unequal distribution of blood in the mucous and cutaneous surfaces, as one part flushes another pales, the same principle ruling as when cold applied to the feet increases the warmth of distant parts, or as when heat applied to the thigh determines a fall in the temperature of the leg or foot of the same side. If these ideas be correct the old and not too much respected dread of the evil results incurred by "driving in" certain eruptions would seem to have good foundation. At least, we may often find it worth while to look at more than the leg which displays the eczema, and to take a comprehensive view of all the conditions to which it may bear relations of sequence, or simultaneity, or alternation. It is very possible in cutaneous medicine to achieve a dangerous success.

On the present occasion I shall draw attention to only one more illustration, that of the baldness which is often associated with anal pruritus. In persons of highly sensitive nervous system a form of local baldness often occurs. Over patches of from one to four square inches the scalp is found depressed, smooth, firm, and hairless. The denudation and shrunkenness of the skin are just like what is seen after *lupus erythematosus*. Possibly the morbid processes are identical. This condition is associated with and long preceded by a harassing anal pruritus. As rectal ascarides are often proclaimed by a certain kind of nasal pruritus, anal pruritus is often proclaimed by this form of baldness, the outcome of a condition altogether different from the condition introductory to eczema. Vaso-motor contraction replaces vaso-motor relaxation, and the starved skin passes through a low wasting inflammatory process to barrenness. In future notes I propose to consider some reflex causes of erythema and *lupus*.

*3. On some reflex influence exerted by the skin on internal organs.*

Dr. Brown-Séquard has pointed out that the ulceration of the duodenum which occurs after severe burns of the skin is best explained upon the supposition that it is a result of reflected irritation. The inflammations of internal organs consequent on exposure of the surface to chill are illustrations of the same principle. There can be no doubt, indeed, that the stimulation of the skin or of parts of it by the atmosphere—by a medium cooler than itself—is a necessary condition of health. The stimulation when kept within due bounds is roborant of the central nervous system, as is generally recognised when we speak of a bracing climate or of a bracing day. What may be the elements of such stimulation cannot be here fully discussed. It is sufficient to recognise the fact that contact with moving air is necessary to the maintenance of a certain vigour of nervous system, and to proceed to the induction that a healthy state of the skin is necessary to a healthy state of the central nervous system. As stimulation carried to excess, in the case of extensive burns, for instance, produces fatal shock, stimulation in default tends equally, I believe, to torpor and languor, the effects of hot baths and hot climates being cases in point. I wish also to argue that torpor of the skin produces or tends to produce torpor or lethargy of the central nervous system. In several instances I have seen a state of inaction of the skin produce a torpor of the nervous system quite unlike the torpor produced by a narcotic. The torpor is not a tendency to sleep, but what I should call a bradæsthesia or slowness of perception and response. Slow thought, slow speech, slow action, with great aptness to weariness and fatigue, to depression by external cold, and with, indeed, little capacity of sleep. It is very possible to argue that such symptoms are due to the operation of morbid poison retained in the system by the failure of the excretory action of the skin. And if by hot baths, particularly by hot-air baths, the skin be roused from torpor and active perspiration induced, the pressing sleeplessness and irritability are at once relieved. That a morbid poison should be removed so quickly is not probable, seeing that poisons in general require a considerable period of

time for their elimination, particularly when their physiological effects have been well developed, for poisons which are operating on the elements of tissues, outside the blood current, are only slowly shaken off and taken back into the current. In the cases to which I am now referring, however, a sense of relief and comfort, a sense of the removal of a local source of irritation, of the relaxation of a distressing tension, is felt directly the skin is made to act, and lasts so long as the action lasts.

And this leads me on to the subject which I had chiefly in view when I began this note, a subject which perhaps ought not in logical strictness to be treated of under the heading of "Nervous Disorder." It is the influence of baths in reducing high bodily temperatures. Now, when a patient having a bodily temperature of 105° Fahr. is cooled down to 95° or lower by immersion for a time in water at 40° or 50°, in iced water, as is recommended by Dr. Routh and others, the phenomenon is not uncommonly included by the observers among ordinary physical phenomena. As a heated bar of iron is cooled by dipping in cold water, so the heated human body. But I have heard physicists urge in opposition to this view that the body cools under the conditions mentioned more rapidly than a similar bulk of water or hydrated colloid would cool; and without going into the whole of this difficult question in physics, though one might soon settle the matter by a few experiments in the dead-house, I wish to point out that the nervous system as well as the circulating system plays a part of the greatest importance in the whole phenomenon. What I mean will be best suggested by the following note:—A medical man, once a student of this hospital, asked me one day in summer to see one of his children, a boy of two and a half years. The child had in the spring had a bad attack of diphtheria. Mr. Parker, then house-surgeon of the Children's Hospital, Great Ormond Street, had performed tracheotomy, and success had rewarded his skill so far as the preservation of the little patient's life was concerned. There was left, indeed, a singular difficulty, namely, that whenever the tube was removed a violent access of feverishness occurred, with rapidly consecutive catarrh affecting the smaller bronchi; therefore the tube had been replaced and continued to be worn. The cause of all this trouble was held to be a paralysis of laryngeal muscles.

A few days before I saw the child the tube had been once more removed, and the usual effects had followed. For two days the temperature had ranged between  $98^{\circ}$  and  $105^{\circ} 2^{\circ}$ . The tube had been replaced, but, after a day of calm, severe capillary bronchial catarrh had set in, and the temperature had run up to  $104^{\circ} 5^{\circ}$  at the time of my visit. The pulse was 160 and upwards, the respiration 70 in the minute. It was clear that there was pressing danger; the most appropriate internal remedies, and all ordinary external applications, though in full use, were clearly overpowered by the increasing mischief. The child was desperate in his restlessness and struggle for breath. On consultation with the father and Mr. Parker, it was resolved that a graduated bath should be tried. The child was placed in a bath at  $100^{\circ}$ , and the bath was cooled in half an hour's time to  $90^{\circ}$ . At the end of that time the bodily temperature had fallen to  $102^{\circ} 5^{\circ}$ , and the chest symptoms were much relieved. The next day the temperature, pulse, and respiration rose again, and at 11 a.m.  $105^{\circ}$  was reached. The bath was used again. Under its use all the symptoms yielded. The temperature fell to  $99^{\circ}$ , the pulse from 150 to 120, the respiration from 72 to 42. For two days after this the temperature remained below  $100^{\circ}$ , but on the next a fresh outbreak of the catarrh took the temperature up to  $104^{\circ} 4^{\circ}$ , the respiration to 52, and the pulse to 136. I saw the child in the afternoon, and finding that all the symptoms were showing hourly aggravation, recommended the use of the bath for the third time, and waited to see the effect. The child was, before the bath, in a state of piteous distress. The breathing was difficult, rapid—now over 70—and attended with loud wheezing and rattling noises. Nothing could be heard but a mixture of fine crepitant with noisy rhonchi and wheezings all over the chest. The face was anxious, the lips livid, the power of speaking lost. There had been no sleep for many hours. The child, weary with its long struggle, was constantly laying its head down, and was inevitably compelled the next moment to raise it again in order to keep on breathing.

A bath was at once prepared, and when the required temperature of  $100^{\circ}$  had been obtained the child was placed in it. Within ten minutes the severity of the symptoms began to remit. At the end of the half hour, the bath being now at  $90^{\circ}$ ,

the axillary temperature was 99°, the breathing was 36, and there was little noise accompanying it. Before the child could be dried after coming out of the bath he fell asleep, and on awaking was breathing with comparative freedom. In the evening the pulse was 108, the respiration 32, the temperature falling. The catarrh had nearly disappeared. Three days after the child was well enough to go to Margate and has ever since done well.

In watching this case I was more impressed by the remission of the bronchial congestion than even by the fall of temperature; and this, conjoined with the removal of the painful unrest, with the smoothing-out of the lines of agony from the little face, seemed to me distinctly related with a soothing influence exerted on peripheral nerves and propagated thence to central and vaso-motor nervous system. It was only a few minutes after the application of the water to the whole surface of the body that the impediment to respiration began to give way; and not till after this did the temperature begin to fall. The origin of the high temperature was in the respiratory tract, and, the course of remedial influence following the course of morbid influence, the general fever abated on the subsidence of the local exciting cause. We may find here one more illustration of the principle that diseases ought to be treated in relation to their causes rather than to their symptoms, with its important corollary that many symptoms, in themselves of serious nature, are much better combated by treatment related to their origin than by treatment directly opposed to themselves.

A case which came under my care in August, 1875, compares well with the foregoing. In respect of this I wish to record my obligations to Mr. Rossiter, at that time house-physician, first for his unwearied care of the patient, and secondly, for his kindness in preparing for me an excellent history of observations.

A boy of 17 was admitted under my care (in Dr. Bristowe's absence) to Charity Ward. He was in a drowsy condition, with a temperature of 104°, and some indications of bronchial irritation, and it soon became evident that he was suffering from enteric fever, and was in the third week of the disease. The temperature fell after his admission, and on the morning of the third day was 98.5°, rising by evening to 101.4°. Thence it rose

again daily till on the thirteenth day (nearly at the end of the fourth week of the disease) it rose above 105°. Delirium had set in, and the diarrhoea was very considerable.

On the day mentioned (August 31st, 1875) Mr. Rossiter writes :

“Twelve motions in twenty-four hours. At 4 p.m. temperature had risen to 104·8°, and at 5.20 p.m. to 105·2°. A bath (six feet long) was brought to the bedside, filled with water at 100° F. Patient was gently lifted into it, and the water was allowed to cool to 92° in the course of one hour.”

(The subjoined figures show the gradual diminution of the temperature of patient.)

Time.	Pulse.	Temp. (axilla).	Temp. of bath.	Remarks.
5.25 p.m.	—	105·2°	100°	—
5.35	188	—	99·5°	—
5.45	146	—	98·4°	—
5.50	142	101·2°	97·6°	—
6.0	144	101·1°	97·2°	—
6.5	—	—	95·0°	Cold water added.
6.15	156	—	98·0°	—
6.20	—	98·6°	92·0°	Patient taken out of bath.
6.25	148	100°	—	Temp. of ward 69°.
8.25	—	109·5°	—	—
9.30	—	109°	—	—

“The first effect of the bath was to soothe the patient. After twenty minutes he began to be restless, as before immersion, and towards the end more so. Though his temperature was 98·6° and that of the bath 92° his teeth chattered. He was now lifted out of the bath on to the bed and rapidly dried; then wrapped in a blanket and left quiet. He immediately went to sleep and slept soundly for several hours—a thing he had not done for many days. It was now arranged that whenever the temperature was rising above 104° the bath should be repeated.

“On September 3rd (second day of fifth week) accordingly he was placed in the bath for half an hour. The temperature fell to 99°, but rose soon after to 102·4°. He did not sleep afterwards.

“On the 4th and 6th the baths were used. On each occa-

sion the temperature fell rapidly, but rose again towards evening, reaching  $102\cdot4^{\circ}$  on the evening of the 4th, and  $103\cdot6^{\circ}$  on the following morning."

Mr. Rossiter writes on September 9th:—"Since the last bath his temperature has gradually sunk, the diarrhoea ceasing (now only two or three motions daily), and general appearance improving. Now sleeps quietly. Pulse 120, soft, dicrotous." A thin, slightly-formed boy originally, he had now wasted to an extraordinary degree, and it was difficult to believe that he could rally to recovery.

In the succeeding week he caused great anxiety, the temperature falling below the normal degree, the lowest point recorded being  $95\cdot2^{\circ}$  on the 13th September. Bedsores formed in October, and went so far as to expose the sacrum; but after a tedious convalescence he was well enough in March to go to Bognor. During the summer he came to see the sister of the ward, who reports him as looking healthy and rather plump. Although the history does not record it, I may state that before his third bath he had sharp bronchial catarrh, and that having ordered the bath to be given, notwithstanding, on the temperature rising to the point fixed, I had the satisfaction of finding this symptom yield with others. The catarrh returned afterwards, but with no great severity.

The axillary temperature was always taken in this case because of the impossibility of getting observations in mouth or rectum by reason that the patient was delirious, and was suffering from sharp diarrhoea. It is very probable that the general temperature did not fall so rapidly as the temperatures registered would indicate. But it is clear that the upward movement of the temperature was on each occasion checked; and a more moderate heat was found prevailing for a varying length of time afterwards. In this respect the use of the bath cannot be denied, in that it arrested the system on a dangerous, if not fatal, course. The reflex influences producing sleep, diminishing diarrhoea, and checking the catarrh, help one to understand the way in which the general relief is brought about, namely, by reflection of soothing influence to the irritated bowel, around and about the ulcers—a reflection in which, again, vaso-motor nerves bear probably a prominent part. I have thought that, pace *Æsopi*, the same remedy might have been used with some

modification when the temperature fell so low ; for it is but a question of degree, and the use of a hotter bath would naturally tend to revive action, as a cooler one had checked it.

In answer to which consideration the quotation of two cases is appropriate. The first is that of a young man, aged 21, who was under Dr. Bristowe in Charity ward with angular curvature of the spine and paraplegia. The notes are here again supplied by my friend Mr. Rossiter.

"The patient had angular curvature in the middle and lower dorsal region since the age of four, when he is said to have injured himself by falling off a gate. Fifteen months ago, after getting wet, he found the back become painful. Four weeks ago the abdomen and legs began to lose feeling, and paralysis of motor power slowly followed.

"*On admission.*—The curvature has its greatest projection at the spines of the sixth and seventh dorsal vertebrae. He is unable to move legs or feet. If the foot is tickled he feels it and involuntary reflex movement is produced. There is now neither pain nor tenderness over the spine, and no hyperæsthesia anywhere."

At the end of five months (in August, 1875) the legs were much wasted, but sensation was good, there was no hyperæsthesia, and the reflex movement was active. It appeared to me probable that in consequence of the curvature the anterior columns of the cord were dragged or pressed against the bodies of the vertebrae, and that they were, therefore, not well supplied with blood. It was to be inferred that the cord was not irreparably damaged or actively inflamed, from the absence of local tenderness, of pain, and of over-acute sensation, and from the presence of natural sensation ; and I thought that if I could apply such stimulation as should send a stronger arterial current into the cord enough activity might be set up to bring him to his condition of nineteen months before. Faradic currents had been tried without effect, so now continuous currents were used. They failed also, and no more good was got out of spinal ice bags. At length it occurred to me that the cord might be roused by an extended stimulation of the skin.

The patient was ordered to be placed in a bath at 100° Fahr. for twenty minutes daily, with immersion up to the curvature. After two baths the patient said that "he felt life coming into

his legs." It was evident that there was a remarkable change in the capillary circulation ; for whereas before the baths the skin was cold, uniformly pale and flaccid, it had become already warm, mottled as in a healthy infant and firmer. The continuous current was now used after each bath from the spine down both legs, with frequent reversal of the flow.

After the fourth bath he was able to move his toes. In a fortnight he could raise his legs from the bed. His recovery went on steadily till he came to walk with the aid of crutches. He was discharged at Christmas, and presented himself at the out-patient room in February, able to stand though not to walk without the support of a stick.

His recovery was certainly aided by his own good will. He was even when paralysed as cheerful and happy as Mark Tapley himself ; in fact, we gave him that name, for he was merry under the most depressing circumstances, and it seemed to us that he richly deserved to get well as he did.

The subject of the second note was a boy of four years and a half, sent to me by Dr. Rhys Williams, of Bethlem Royal Hospital, in June, 1871.

Born healthy, he had taken to his feet early, and walked well at fourteen months. At twenty-eight months he "began to go off his feet," but walked again at thirty-two months. At the end of six weeks he again lost power and, with the exception that ten months ago he walked painfully for a few days, he had been paralysed in the lower limbs ever since.

When I saw him first the legs were much wasted. He was unable to move his legs at all when lying down ; could swing them when sitting, but could not stand. He complained of much pain when the legs or feet were touched, but not when the thighs were touched. Ordinary sensation appeared perfect. The flexors of the thighs and extensors of the leg were powerless, the flexors of the leg retained some power, and it was by these that he accomplished the swinging of the legs. The trunk was well developed, the shoulder-muscles firm and large. There was no distortion of the spine, and no tenderness over any part of it. There was no evidence of joint-disease anywhere. The child was pale, heavy-eyed, and of extremely irritable temper. The head was large, square of shape, with a great expanse of prominent forehead. All the first set of teeth were cut

before the illness began. The teeth were well formed, but the molars showed signs of decay. The child was said to sleep well; had not any facial paralysis, and had no difficulty in urination. The appetite was bad; he refused all meats and even eggs, but would take milk freely. The contrast between the well-nourished and vigorous trunk and upper limbs and the withered lower limbs was very striking. In eight well-marked cases of infantile paralysis which have come under my notice the same sort of contrast has existed. So that the idea of an unequal distribution of nutrition in which the brain and upper part of the spinal cord are well treated at the expense of the lower part of the cord may be entertained. I classed this case at the time among cases of infantile paralysis; for though there was not the more usual limitation to one limb or one group of muscles, there was the same history of loss of muscular power with rapid wasting of tissue, unaccompanied by loss of sensation, as we find in infantile paralysis. There was no distortion of limbs because the paralysis was so general, and the hyperæsthesia was probably caused by the large extent of spinal area involved. Judging that the lumbar enlargement of the spinal cord was probably the seat of some atrophic process, or of some ineffective circulatory action tending to produce atrophy of the cells, I endeavoured, as in the preceding case, to set a better circulation going by stimulation of the skin. As there was no tenderness over the spine, I hoped that the hyperæsthesia was but another sign of defective nutrition, but I proceeded with caution.

The application of a douche at  $105^{\circ}$  to the lumbar and dorsal region of the spine was ordered. It was commenced gradually, the temperature being at first only  $100^{\circ}$  and the first application, was only for five minutes. After a time water at  $110^{\circ}$  was used for ten minutes or more.

It was arranged that the child should take a quart of good milk daily, and the hypophosphite of soda with cod-liver oil were prescribed.

At the end of a fortnight the hyperæsthesia had nearly disappeared, except from the soles of the feet; the child, lying on his back, could now kick his heels in the air. At the end of a month he began to crawl, at seven weeks he stood, and on the 1st of September he walked into my room, without any remaining sign of over-sensibility.

A year later when I heard of him he was running, walking, eating, and sleeping like any other child of his age, and was showing good intellectual power.

In these two instances we have before us the agency of extensive stimulation of the skin in rousing a portion of the central nervous system from a dormant state, from a state tending to wasting of the function-sustaining tissue. The cases bring out the middle term involved when uterine haemorrhages or leucorrhœas are checked by the application of hot douches to the dorsum, or when an epistaxis is checked by the familiar cold key thrust down the back of the neck. They illustrate the possibility of rousing reflex trophic influence in wasting limbs by stimulation of a dormant central ganglion through the peripheric nerves; and second the possibility of restoring the dormant central organ to permanent natural functional activity by long continuance of the peripheric stimulation.



## ON CLEFT PALATE.

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IN the last volume of the 'St. Thomas's Hospital Reports,' for 1875, I contributed an article on Harelip. In discussing that subject it would have been no difficult task to extend the paper to undue length by including the frequently associated condition of cleft palate. But it appeared to me that the latter deformity was of equal importance, and that to do justice to the subject it would be a better plan to consider it in a separate communication. Hence this contribution. Such an arrangement has at least this advantage, that, by increased opportunities at the hospital and elsewhere, I have been enabled to acquire a more mature experience of the malformation of cleft palate, and am, therefore, in a better position to estimate the relative merits of the operations that have from time to time been devised to effect union of the fissured parts.

As this paper is intended to be a supplement to that on harelip, it will be most convenient to consider the subject of cleft palate much in the same order. Whilst many of the points to which reference has already been made must necessarily be again touched upon, every effort will be used to avoid, as far as possible, needless repetition. Further, whilst I shall briefly allude to the treatment of perforations of the palate the result of accident or disease, the principal object of the present communication is to review the subject of congenital mal-

formations, the different operative procedures being more particularly dwelt upon.

Congenital fissures of the palate assume a variety of forms. Thus, in one case the split will extend through the uvula only (fig. 1). In another it will involve more or less of the soft palate, stopping short at the margin of the palate bones (fig. 2).

FIG. 1.



FIG. 2.



In a third the fissure will include a portion of or even the whole of the hard palate (fig. 3). In other examples, in which the deformity is complicated with harelip, whether single

FIG. 3.

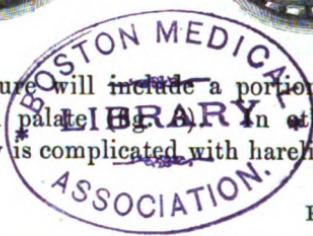


FIG. 4.



or double, the alveolus in front will be more or less involved (fig. 4). On examining the roof of the mouth in these cases the vomer may in some examples be seen to be placed free and

exactly in the median line, so that a probe may be passed into the nasal cavity of either side (figs. 4 and 5); whilst in other instances the septum is attached to one or other half of the palate, thus shutting off the cavity of the nares of that side from the buccal cavity (fig. 6). Referring to this point, Rouge<sup>1</sup> states that the vomer is most frequently attached to the right side. Lastly

FIG. 5.

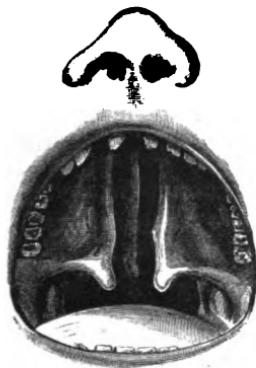
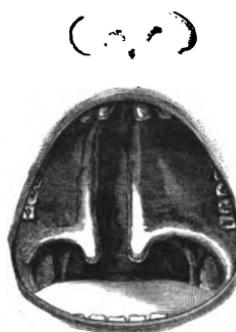


FIG. 6.



there may be fissures of variable size extending through the alveolus (fig. 7); this latter condition may be associated with a cleft of the soft palate only, the rest of the hard palate being to all appearance perfect and arching across like a bridge, as in a case under my care at the hospital in the summer of 1874.

In very exceptional cases there is a congenital aperture in the soft palate without any division of the uvula or palate bones. I have myself never met with an instance of the kind, and the condition must be regarded as rare when Dieffenbach declares he has only seen one example, and this occurred in a young medical student.<sup>2</sup>

Trélat, Notta, and Langenbeck refer to cases in which the hard palate has been deficient, the gap being filled in by the mucous membrane only, stretched from side to side.

Whatever the extent of fissure, there is generally more or less of the hard and soft palate observable on each side, yet

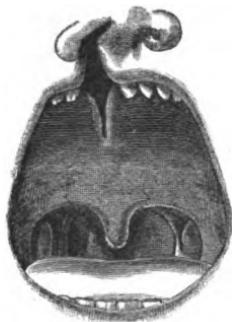
<sup>1</sup> Rouge, 'L'Uranoplastie et les Divisions congenitales du Palais.'

<sup>2</sup> 'Die Operative Chirurgie,' von Johann Friedrich Dieffenbach, Erster Band, 1845.

there are even exceptions to this rule ; thus, M. Ancelet<sup>1</sup> refers to an example in which there was a *total absence* of the soft palate in a child ; and amongst other anomalous conditions, an instance is reported in the 'Brit. Med. Journal' for March, 1857, in which the left side of the uvula adhered to the edge of the soft palate near the tonsil.

Besides the fissured palate other deformities have been occasionally met with in the same patient. I remember one curious instance which was under the care of Mr. Bowman at King's College Hospital in 1859. There was eversion of the lower lip with two openings of buccal glands, besides a complete fissure of the palate with double harelip. These fistulous openings perpetually discharged a secretion, which so annoyed the patient that an operation was required. It consisted in dissecting

FIG. 7.



up the apertures and turning them back so that the discharge might enter the cavity of the mouth.<sup>2</sup> And in a case of complete cleft of the palate sent to me by my friend Mr. Samuel Osborn there was also a congenital fissure of the lobe of the right ear, in another example congenital talipes calcaneus was present in both feet, and in a third the little patient had marked hypospadias.

In looking into a patient's mouth it is well to bear in mind that sometimes the appearance after an injury of the upper jaw closely resembles that after an operation for cleft palate. I was

<sup>1</sup> 'Bullet. Méd. de l'Aisne,' 1867, No. 2.

<sup>2</sup> 'Brit. Med. Journ.,' August 20th, 1859, p. 666. This case is probably somewhat similar to the one to which I referred in 'St. Thomas's Hospital Reports,' 1875, p. 141, art. "Harelip."

much struck with this fact in the case of a man aged 19, who applied as an out-patient at St. Thomas's Hospital on June 1st, 1876, for another ailment. The right side of his face was peculiar, which led me to examine his mouth. He stated that when a boy he was kicked on the right cheek by a horse. On carefully examining him the lateral incisor tooth of the right side was absent, and there was a deep V-shaped furrow extending from before backwards. There was no distinct fissure, but in the absence of any history it might easily have been taken for a case in which a successful operation for cleft palate had been performed. It showed, at least, that injuries sometimes occasion deformities that have a somewhat similar appearance to those of a congenital origin.

The main difference between cleft palate and harelip is that the former is always in the median line, whilst in harelip the fissure is, as a rule, on one or both sides. Cleft palate, as Velpeau says, has never yet been seen double. Harelip attracts the attention of bystanders, a cleft palate does not offend the eye in the same manner, but when the patient speaks the ear at once detects the malformation. It is scarcely possible to estimate the number of adults who are practically excluded from society by this distressing deformity. There is no doubt that many infants with fissured palate die very early of sheer starvation. They are unable to suck, and if food be administered by the spoon so much returns through the nose that a sufficient quantity is not swallowed to insure nutrition. At my suggestion Messrs. Maw and Co. have manufactured an instrument such as this. It consists of an ordinary teat attached to a feeding bottle.

FIG. 8.



Over the teat is a very thin plate of soft metal, which can be moulded to the little patient's mouth. The instrument is not available in all cases, but is, as I have reason to know, well worthy of trial. Mr. Oakley Coles has devised a somewhat similar apparatus, but the shield is made of india rubber.<sup>1</sup>

<sup>1</sup> 'Mechanical Treatment of Deformities of the Mouth.'

According to Mr. Atkinson,<sup>1</sup> any such apparatus is scarcely needed, for he declares that "when a child with a defective palate sucks the breast it places the nipple under the tongue, and thus instinctively makes an artificial palate of its tongue which prevents the milk from flowing into the nose instead of the pharynx." There is no doubt that the little patient should, when fed, be placed in the almost upright posture, and that mother's milk should be given, either from the mother herself or from a wet nurse. I have, however, noticed that many children with congenital cleft palate appear to thrive better under a more farinaceous diet.

When the patient arrives at boyhood or girlhood the regurgitation of food through the nostrils does not frequently occur, but some care has to be exercised in order to prevent it from doing so. In cases of complete cleft through the hard palate there is often a deficiency in the sense of smell.

Some writers, Mettauer amongst others,<sup>2</sup> speak of extreme fœtor of the breath. I cannot say I have noticed this. There is a peculiar odour which may possibly arise from the mucus becoming rapidly dry and thus forming incrustations on various parts of the mucous surface.

It is further very difficult, and in some cases impossible, for the patients to blow out a candle, and on the same principle they cannot perform on a wind instrument, such as the flute or cornet. M. Roux noticed these points in his first operation on Mr. Stevenson.

It would be foreign to the purport of this paper, which is intended to be a practical one, were I to enter into the consideration of the development of the mouth. For minute and elaborate descriptions of this the reader is referred amongst others to M. Coste's<sup>3</sup> excellent work, to Mr. Goodsir's exhaustive article,<sup>4</sup> and to an excellent résumé by Dr. T. Hamy.<sup>5</sup>

It is, however, not difficult to find the explanation of the origin of fissures of the lip and palate when it is remembered

<sup>1</sup> 'Lancet,' 1838, vol. i.

<sup>2</sup> 'American Journ. of Med. Science,' vol. xxi, 1837-38.

<sup>3</sup> 'Histoire générale en particulière du Développement des Corps organisés.'

<sup>4</sup> 'Edinburgh Medical and Surgical Journal,' vol. li.

<sup>5</sup> 'L'Os Intermaxillaire de l'homme à l'état normal et pathologique,' 1868, Paris.

that in normal development at an early period of foetal life the nose and mouth form one common cavity. At about the eighth or ninth week the horizontal plates of the superior maxilla of each side unite in the median line, and also with the incisive bone; and further, that from the superior maxillary protuberances the palate and superior maxillary bones are developed. Thus, various degrees of fissure, either of the hard or soft palate, will result from non-union of these parts.

MM. Follin and Duplay<sup>1</sup> put the case very plainly when, after giving an elaborate account of M. Coste's views on development, they state, "It is easy to understand all the complications of harelip and cleft palate if we suppose that the arrest of development extends to the bones and the soft parts. If the separation is persistent between the superior maxilla and intermaxillary bones there may be a labio-alveolar fissure, and if it entered further back it will be labio-palatine. And, again, if the arrest of development occur very early in foetal life, when the nasal and buccal cavities freely communicate, the deformity known as a complete cleft palate will be the consequence." As bearing on development I may direct attention to an able paper by Dr. Langdon Down on "The Relation of the Teeth and Mouth to Mental Development."<sup>2</sup> He says:

"I have made a very large number of careful measurements of the mouths of the congenitally feeble-minded and of intelligent persons of the same age, with the result of indicating, with few exceptions, a markedly diminished width between the posterior bicuspids of the two sides. One result, or rather one accompaniment, of this narrowing is the inordinate vaulting of the palate. The palate assumes a roof-like form. Often there is an antero-posterior sulcus corresponding to the line of approximation of the two palate bones. There is very frequently a deficiency in the posterior part of the hard palate, from a want of development of the palatal processes of the maxillary bone, as well as an absence of the palatal process of the palate bone. As a result of this defect the false palate hangs down abnormally and interferes with clear phonation." Dr. Down then adds that at an early period of his investigations he was prepared to find a large number of cases of cleft palate, but he discovered by

<sup>1</sup> 'Traité élémentaire de Pathologie externe,' tome iv, fascicule 3, p. 645.

<sup>2</sup> 'Trans. Odontological Soc.,' vol. iv, 1872.

statistics that these were not more than five in one thousand cases. Bisection of the uvula occurred four times in one thousand, and absence of the uvula twice. The excessive vaulting of the palate, he adds, may possibly arise from arrest of development of the sphenoid bone or defective growth of the vomer.

I have not been enabled to ascertain the percentage of cases of harelip and cleft palate in this country, but the subjoined statistics may be briefly referred to, so that the reader may form some idea of the proportion of such cases on the Continent. According to Grenser, of 14,466 infants born living at the Maternity of Dresden from 1816 to 1864, there were sixteen cases of simple harelip and nine with fissures of the palate. And Credé states that from October 1st, 1856, to December 31st, 1865, 2044 children were born. Out of this number there was one case of simple harelip and one of complete division of hard and soft palate.<sup>1</sup>

It is somewhat curious that the notion of closing a fissure of the palate by operation is only of comparatively modern date, principally, indeed, within the present century. As Sir William Fergusson truly remarks, "The early history of the operation for cleft palate sounds like a romance."<sup>2</sup> And in order to show how little operative interference was considered justifiable there is, I observe, no mention made of the subject in Cooper's 'Surgical Dictionary,' published in 1818. And Sir Astley Cooper, writing in 1823, implies that little can be done for the deformity.

Sir William Lawrence, too, speaking in 1829,<sup>3</sup> says that "there are few cases in which the operation is required."

Further, Mr. Syme, writing so recently even as 1854,<sup>4</sup> believed the operation was of doubtful expediency, and states, in his 'Principles of Surgery,'<sup>5</sup> "Split palate does not admit of any remedy for the division of the hard palate, except the closure of the communication between the nose and mouth by a piece of silver, enamel, or other substance so fitted as to retain it without shifting. Fissure of the soft palate may be

<sup>1</sup> Rouge, *op. cit.*

<sup>2</sup> 'Lectures on the Progress of Anatomy and Surgery,' 1867.

<sup>3</sup> 'Lancet,' vol. ii, p. 959.

<sup>4</sup> 'Association Med. Journ.,' March 10th, p. 280.

<sup>5</sup> 'Principles of Surgery,' 1856, 4th edit.

united in favorable cases by an operation similar to that for harelip, but which is uncertain of execution owing to the situation of the parts, their mobility, and the involuntary efforts of the patient." How vastly different is our experience of to-day!

As to the *hereditary character* of cleft palate it does not seem, if compared with harelip, to descend so frequently from parent to child. In most of the cases that I have observed there has been no history, and yet in the few exceptions the circumstances have been somewhat striking. Thus, at St. Thomas's Hospital, in 1874, I saw two children, sisters; one had a simple cleft in the soft palate, and the other a double harelip with a complete fissure of the hard and soft palate. The father and mother showed no similar condition, but the mother's aunt had a fissure of the palate. In another instance the father and child had cleft palate, and there were three other children by the same father whose palates were perfectly normal. In another instance a father and two children all had cleft palate, and one child had harelip besides.

Rouge<sup>1</sup> refers to the cases of two sisters with cleft palate, one rather worse than the other. The parents were perfectly healthy, and there were two brothers normally developed, but otherwise there was no trace of similar deformity in the family.

Other examples might be adduced of a somewhat similar nature. Mr. Ramsay, who read a paper at the Odontological Society in 1865, stated his belief that the deformity was not hereditary, and further mentions a point which is certainly at variance with my own experience—that he had never seen a case in which the patient's complexion was dark. It is a matter of little moment, but I am inclined to think that most of the patients I have seen have had a somewhat nervous temperament, and many, perhaps the majority, have had fair complexions.

As to the *causes* of cleft palate, I have observed that parents are not so ready to supply a reason for that deformity as they are in cases of harelip. Maternal impressions of all sorts are of course given. Thus, in one case now under my observation the mother attributes the deformity to the fact that when she was pregnant one of her other children fell on a walking stick

<sup>1</sup> Op. cit.

and split open the soft palate. In another the mother said it was due, she believed, to her having longed for some particular fish which had a huge mouth. What the fish was I could not ascertain. She assures me that this occurred at about the sixth week of gestation.

The merit of having first performed the operation has been claimed both by Professor Graëse, who published his unsuccessful case in 'Hufeland's Journal' in 1816, and by M. Roux. M. Roux certainly seems to have been the first to excite the interest of the profession to the subject, for he operated in September, 1819, and according to his own showing at least acted perfectly independently. Thus he says, "Je declare, sur l'honneur, que jamais rien ne s'etait offert à mon pensée, et que je n'avais reçu non plus aucune inspiration étrangère relativement à la suture de voile du palais, lorsque je fus conduit à entreprendre cette opération sur le jeune médecin du Canada" (Mr. Stevenson). This case is fully given in his 'Mémoire sur la Staphylorophie,' Paris, 1825.

But about the same period (1820) Dr. John C. Warren, of Boston, performed an operation for closing the soft palate. Thus he says, in the 'American Journal of the Medical Sciences,' vol. iii, 1828, "Some years ago I had occasion to perform an operation for remedying the natural fissure in the soft palate. At that time I understood the operation had been once done in Poland or Germany, and once by Professor Roux, but I sought in vain for details that might assist me in its performance. However, I executed it satisfactorily then, and have since repeated it." He then gives an account of his first operation—which was successful—on a young woman aged sixteen.

The operation had, however, been successfully performed previously, for, according to M. Robert,<sup>1</sup> M. Lemonnier, a dentist, succeeded in uniting the two borders of the cleft in the case of a child about the year 1760. He first inserted several points of sutures in order to keep them approximated, and afterwards abraded them with a cutting instrument. Upon which Velpeau observes, "A child, a cleft, the suture, the refreshing, the cure, everything, in spite of the somewhat vague expressions of Robert, scarcely permit us to doubt that this dentist truly had recourse to staphy-

<sup>1</sup> 'Mémoires sur différents objets de Médecine,' Paris, 1764.

loraphy and not to suture of a simple perforation of the palatine vault."<sup>1</sup> Eustache (of Beziers) is said to have performed the operation in 1770, and in 1801<sup>2</sup> Désault reports the case of a child in which the palate was closed twenty-seven days after the operation for harelip. In 1813 M. Colombe attempted the operation on the dead body, and failed to induce a living patient to submit to the proceeding in a suitable case in 1815. Dr. Stevens, of New York, and Mr. Mettauer performed the operation in 1827.

It must be remembered that all the above cases were fissures of the soft palate, for no one had thought of closing the hard palate. It is supposed by Rouge that to Krimer is due the credit of having been the first to close a fissure of the hard palate, which he did in 1824, in the case of a girl aged eighteen.<sup>3</sup>

Other surgeons immediately followed suit, and undertook operations for closing both the hard and soft palate, amongst others Dieffenbach, Mason Warren, Liston, Alcock, Brodie, Guthrie, Bushe, and Crampton; and more recently Avery, Pollock, Sir W. Fergusson, the last distinguished surgeon's experience having, perhaps, surpassed any of the others.

It is somewhat remarkable, that though assumedly the first case (that by Lemonnier) was in an infant, and the success perfect, yet to within even a few years it has been considered the correct practice to defer the operation until the patient has arrived well nigh to the age of puberty. Velpeau has expressed this opinion, and Müller thought that from the sixteenth to the eighteenth year is the best time. Within even the last twenty years the same theory has been held by some surgeons; and as an illustration I may say that at St. Thomas's Hospital last year (1876) a boy, aged sixteen, applied to me to be operated on, and stated that his mother had been told that the operation could not be safely undertaken until he was sixteen years of age. He certainly took the earliest opportunity of seeking advice, for he applied on the anniversary of his birthday. Further, M. Velpeau<sup>4</sup> thus expresses his opinion on this point:—“Toutefois, l'opération échoue encore assez souvent; une jeune

<sup>1</sup> South's 'Chelius,' vol. i, p. 603, 1845.

<sup>2</sup> 'Œuvres chirurgicales,' p. 204.

<sup>3</sup> Op. cit.

<sup>4</sup> 'Méd. Opératoire,' 2nd edit., vol. iii, p. 561.

fille que j'ai vue à l'Hotel Dieu l'a subie cinq fois sans succès. Plusieurs malades de M. Roux en sont morts, et j'en ai vus un certain nombre que ce chirurgien avait opérés sans fruit. Les autres praticiens ont obtenu une proportion de résultats heureux moins forte que M. Roux, en sorte qu'il n'est prudent de la tenter que dans les bonnes conditions, chez des sujets bien portants, dociles, et âgés de quinze à cinquante ans, par exemple."

However, within the last fifteen years, surgeons have practised the operation at a much earlier period of life; thus, Billroth operated in 1859 on a child six months old at the same time he operated on the harelip. The harelip united, but the palate failed.<sup>1</sup> He also operated in 1861, successfully closing the hard and soft palate at the same time in a child aged about two and a half, and with partial success in a child aged one year, complete failure in a boy aged eight weeks and also in a girl one and a half year old.<sup>2</sup>

Otto Weber operated in 1861 on a child six weeks old; the soft palate failed, the hard united. From 1863 to 1865 M. Gustave Simon operated on three cases, one child five days old; hard and soft closed at same time; hard palate united, soft failed. Second case, child nine months, good result. In this case the harelip and palate were both done at same time. Third case, a child aged twenty weeks, failure. And another case, aged six days, of uraniscoplasty, the child died eight days after of diarrhoea.

M. Ehrmann, writing in 1870,<sup>3</sup> reports five instances of fissured palate. The patients were aged respectively three and a half years, four and a half months, eight months, eight weeks, and twenty-seven months. And Mr. Marsh operated on a case with partial success at the age of sixteen months,<sup>4</sup> and I operated successfully on a child at St. Thomas's Hospital, aged two months.<sup>5</sup>

Of Mr. T. Smith's eleven cases included in a paper published in the 'Transactions' of the Royal Med. and Chir. Soc. for 1868, the eldest was twenty-seven and the youngest two years of age.

<sup>1</sup> 'Lancet,' 1852, vol. ii, p. 31.

<sup>2</sup> 'Archiv. f. Klin. Chir.,' 1862, t. xi, p. 658.

<sup>3</sup> 'Lancet,' August 20th, 1870, p. 259.

<sup>4</sup> 'Brit. Med. Journ.,' November 6th, 1869, p. 520.

<sup>5</sup> *Ibid.*, January 6th, 1872, p. 15.

M. Gustave Simon held that the operation ought to be performed during the first six months of life, and by preference during the first or second. A greater number of patients die, he says, after the operation, but in those that live the result is much more satisfactory. The muco-periosteal flaps are also said to be more readily stripped off in infants than in adults.

In expressing my own opinion I have no hesitation in stating that operations on very young children are, as a rule, extremely unsatisfactory, and this is the experience of other surgeons; thus, M. Passavant operated on five children varying from six weeks to two and a half years without one success. Langenbeck operated at five months and two and a half years without a better result. Billroth operated on a child two months old, who died six hours after the operation, and Rouge operated on a child six weeks old, who died twelve days after staphyloraphy.

The surgeon must, of course, be guided by the peculiarities of each case. I have myself operated on very young children in several cases, and, as just stated, one child was two months old; but this case was a very favorable one, for the fissure only involved the soft palate. I am, however, inclined to think that unless there be good reasons for doing it, the operation should not be undertaken before the age of five or six. Langenbeck advises staphyloraphy "not under seven years." Any one interested in the subject has only to look at the fissured palate in a newly born infant, and he will see how extremely thin the mucous lining is. It is almost like tissue paper, which with the slightest touch of the finger-nail will break away. I myself cannot conceive that operative measures under such circumstances can be of much avail. If the case be watched (and I have now some twelve or more cases under my personal observation), the gradual and slow development of the soft palate, as well as the covering of the hard palate, into a tough, thick, and solid structure may be readily observed. Chloroform necessarily is a great boon during the operation, but in very young children the chief difficulties arise in the after-treatment of the case. From sheer ignorance the little patients are apt to do something that promotes disunion; perhaps they will cry perpetually, or cough, or sneeze, or play with the ligatures with their tongue, and such acts favour the separation of the parts.

I would here mention that if the wound bursts open, the surgeon should never despair of getting considerable, if not complete union, provided the smallest portion of the edges can be got to adhere. The persevering application of strong nitric acid will promote granulations, and I have seen surprising results in cases which I at first regarded as hopeless. I am especially reminded of one case, that of a boy, aged four, upon whom I operated rather more than two years ago, and who was going on quite well until one day he gave an unlucky cough. The whole of the soft palate gave way, but by using the nitric acid a most perfect cure was effected.

In describing the various operations, it will be convenient to divide them into two classes:

1st. Including those cases in which the soft palate alone is involved. This operation is termed *staphyloraphy* (*σταφυλή*, the uvula, *ῥαφη*, a seam).

2nd. Including those cases in which the hard palate is more or less implicated. These may be remedied by two modes of procedure:—(a) By stripping off the soft tissues from the hard palate (in one or more ways), and so closing the aperture. This operation is termed *uraniscoplasty* (*οὐρανίσκος*, palate, *πλασσω*, I form. (b) By completely dividing the bone and so uniting the sides of the fissure. This operation is called *osteoplasty* (*οστέον*, bone, *πλασσω*, I form).

### 1. *Staphyloraphy.*

Before proceeding with this subject it is necessary to make a passing allusion to the numerous mechanical appliances that have been employed in cases in which the patients have either objected to, or the condition of whose palate has rendered it not amenable to surgical treatment. The practical surgeon is aware that the best constructed apparatus cannot take the place of operative procedure. Most of the instruments that have been suggested have had for their object the closure of holes or perforations, especially of the hard palate acquired by accident or disease. Such instruments are termed *obturators*, and to these I shall presently refer. Various mechanical means or false palates have been employed by Stearns,<sup>1</sup> Kingsley, Sercombe,

<sup>1</sup> 'Lancet,' July 5th, 1845.

Ramsay, and Oakley Coles, as a complete substitute for an operation on the soft palate. In a patient who was exhibited at a meeting of the Royal Medical and Chirurgical Society, November 27th, 1866, "the instrument consisted of a piece of hard vulcanite with two teeth attached to the anterior portion. This supplied most accurately the deficiency in the hard palate. The fissure in the soft palate is closed by means of a piece of soft vulcanite attached to the hard, which is capable of being pressed slightly upwards and downwards by the muscles of the fauces, thus effectually closing the passage of the nares during speech or deglutition."

An ingenious instrument, such as this (fig. 9), has been used

FIG. 9.



by Mr. A. T. Norton in a case of partial cure after operation, a description of which will be found in the 'Med. Press and Circular' of April 19th, 1876.

Mr. James Salter gives an excellent description of an instrument he has devised for the same purpose;<sup>1</sup> and Mr. George Parkinson, who has had considerable experience in such cases, refers especially to this method of treatment.<sup>2</sup> On the other hand, Mr. William Donald Napier, after numerous trials in such cases, has arrived at the conclusion that the value of mechanical apparatus is very much overrated, and is of opinion that no artificial means should be employed excepting in those cases in which it is not possible to perfect a cure by surgery.

Again, in order to avoid the use of cutting instruments, various means to establish inflammation and thus to produce a raw surface have been suggested. Graëfe used caustic potash, and also sulphuric acid, Ebel advised the tincture of cantharides, and Doniges used a hot iron (A.D. 1823).

Dupuytren, Béclard, and Werneck tried cauterization by

<sup>1</sup> 'Holmes's Surgery,' vol. iv, article "Diseases of Teeth."

<sup>2</sup> 'Lancet,' vol. i, 1867, p. 41.

means of muriatic and sulphuric acid, but the results were not favorable.<sup>1</sup>

During the past year I have been trying the application of strong nitric acid to the fissure, and, I think, with decidedly good results. The only drawback is that the process of cure is somewhat tedious. The *modus operandi*, as I explained in the 'Lancet,' July 29th, 1876, is this:—I first produce a raw surface by carefully applying with a stick (not a glass rod) the Acid. nitric. of sp. gr. 1·500, and in a few days afterwards I use in the same way the Acid. nitric. sp. gr. 1·420 (Ph. Brit.), about twice a week to the part, especially to the fork of the cleft. The merits of this procedure have been put to the test by other surgeons. Thus, Mr. Charles Gaine, of Bath, writes to me under date November 26th, 1876, respecting one case, that "The fissure was nearly closed after eight or ten applications of the Acid. sp. gr. 1·500, and about six of the Acid. Nit. pur." Mr. H. G. Armstrong, too, of the Royal Berks Hospital, Reading, states that in one case in which he applied the treatment he was quite satisfied of considerable improvement.

M. Jules Cloquet, like myself, seems to have been fairly satisfied with this mode of treatment, and in 1855 published an essay entitled 'Mémoire sur une Méthode d'appliquer la Cauterisation aux divisions anormales de certain organes, et spécialement à celle du vois du Palais,' in which cases are given of success after repeated cauterizations.

At the meeting of the Academy of Sciences of Paris of the 21st of May, 1860, a case was brought forward by Professor Benoît, of Montpellier, which had been treated by this method. The child was eleven years old, the soft palate was completely cleft, and all the usual symptoms were present. The treatment lasted nineteen months, with two rather long interruptions. The whole cleft has now united save that of the uvula, and this result was obtained by thirty-three cauterizations, fourteen with the acid nitrate of mercury and nineteen with the solid nitrate of mercury.<sup>2</sup>

Mr. Tyrrell reported a case in which he closed a small congenital aperture of the roof of the mouth (a very rare deformity), situate about the centre, in a girl seventeen years old.

<sup>1</sup> 'Dictionnaire de Médecine et de Chirurgie pratique,' vol. xv, 1836.

<sup>2</sup> 'Lancet,' June 9th, 1860, p. 576.

The hole was only large enough to admit the blunt end of a probe, and it was cured by a few applications of a hot iron.<sup>1</sup>

Before undertaking any operation for closing a fissure of the palate, the surgeon should ascertain, as far as he can, that the patient is in the best possible state of health. Occasionally there is enlargement of the tonsils, and their removal is very desirable, because they hinder the prospect of union. It is a good plan, too, to accustom the parts to the contact of the finger without the patient retching; hence for a few days previously the fauces should be touched three or four times daily with a stick or other suitable substance. M. Ebel insisted on this; and though it might appear to be less necessary at the present day, because chloroform or some other anaesthetic is generally employed, yet I think it is useful in the after-treatment. Alum gargle has been also employed with the idea of diminishing the vascularity of the part. The administration of tonics is necessary in some cases, especially in women who have leucorrhœa or other uterine disturbances. I must confess I have my doubts as to the propriety of purging the patient on the day previous to the operation, for I am inclined to think that it is apt to weaken him and so diminish the chance of union. Again, the effect of a purge in many instances is to give the patient a vigorous appetite, hence he is likely to eat with less care than he otherwise would. I have observed this especially in children about seven or eight years of age. The rule that I generally act upon then is, not to give a purgative unless it appear necessary, and then to administer it on the third day before the operation.

Anæsthesia in some form may be employed, but if used it should be carried to some considerable extent, otherwise, if the patient be in the least conscious, the operator is greatly hampered in his manœuvres. In 1852 a writer in the 'Lancet,' vol. i, p. 118, says, respecting the administration of chloroform and such anaesthetics, "Staphyloraphy is, of course, one of the few operative proceedings where chloroform cannot be used." Long before this period, however, surgeons had removed large tumours of the jaw under the influence of this agent, and there appeared to be little reason why staphyloraphy should not be performed with the patient in a state of unconscious-

<sup>1</sup> 'Lancet,' 1829, vol. i, p. 549.

ness. In 1857 Mr. Field, of Brighton, closed a fissure of the palate under chloroform, and Mr. T. Smith brought forward the advantages of anaesthesia in an interesting paper read before the Royal Medical and Chirurgical Society, January 14th, 1868.

I may say that in selecting the kind of anaesthetic it is well to bear in mind that ether excites the salivary secretion. I therefore prefer chloroform in all operations about the mouth, and am supported in this opinion by Mr. Charles Moss, whose great experience as a chloroformist in such cases enables him to speak with authority.

The operation on the soft palate is sufficiently easy, and may be thus performed :—Although some surgeons prefer the upright posture, there is no doubt that the recumbent position is the best both for the patient and the surgeon. The patient's head can be more readily steadied, and the light directed more completely into his mouth. Under chloroform the patient is very apt to struggle occasionally, hence his movements should be restrained by straps applied in the following manner :—The knees are kept down either by a strap or bandage, which passes under the operating table. Another strap or bandage is fastened to one wrist, say the right, and is then carried under the left thigh of the patient and then secured to his left wrist. These straps, it must be understood, need not be put on too tightly; they are only intended to check movement, and should be applied in such a way as to allow of the patient being turned on his side if necessary, so as to clear the throat in case of vomiting. The mouth should be kept open by a gag of some kind. Surgeons have their own fancies on this point. Mr. T. Smith's ingenious instrument<sup>1</sup> is useful, but the instrument I am in the habit of employing (fig. 10), and which answers the purpose remarkably well, is one that was made for me in 1870, and which has since been slightly modified by Sir William Fergusson.<sup>2</sup> I was not aware, until my attention was directed to the fact by my friend Mr. Alfred Coleman, that he had devised a somewhat similar, but rather more cumbersome instrument to that first made for me.<sup>3</sup>

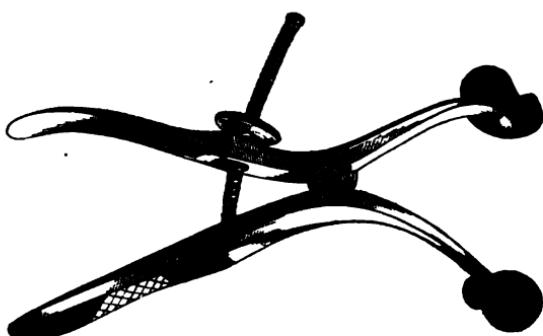
<sup>1</sup> See 'Med.-Chir. Trans.,' 1868.

<sup>2</sup> See 'Brit. Med. Journ.,' January 1st, 1876, p. 8.

<sup>3</sup> See 'Med. Times,' January 26th, 1861.

I am quite convinced that the main difficulty in operations on the palate is the haemorrhage, which is occasionally very trou-

FIG. 10.



blesome; and whilst I think most highly of Sir W. Fergusson's plan of dividing the muscles, yet I am inclined to believe that this part of the operation, inasmuch as it is attended with some bleeding, had better be postponed until after the denudation of the edges. Now that chloroform is so universally administered the operator is enabled to pare the fissure rapidly and generally in one continuous piece, the anæsthetic preventing the sudden contraction of the muscles. The different methods of dividing the muscle will be referred to presently.

The instruments to be employed should be of the simplest character. Roux evidently had a horror of complicated surgical apparatus. He says, after an experience extending over nearly half a century, "Je crains toujours dans la pratique des opérations les instruments qui tiennent trop du jeu des machines. Partout où les actions simples peuvent suffire, c'est de ce côté que sont mes préférences."<sup>1</sup>

The necessary preparations having been made, there should be two or three assistants to hand instruments and to soak up the blood with clean sponges, which latter should be about the size of a walnut. The plan I adopt may be thus described:—The operator, standing on the right side of the patient, commences by seizing with a pair of hook-forceps (fig. 11), a little below the

<sup>1</sup> 'Quarante Années de Pratique Chirurgicale,' t. i, p. 329.

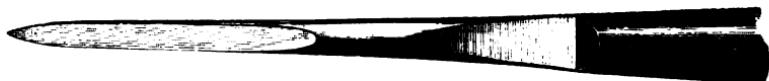
centre of the cleft on the patient's left side. A knife, such as

FIG. 11.



here depicted (fig. 12), is then made to transfix the margin of the cleft, and is carried downwards to the extreme point of the uvula.

FIG. 12.



The instrument being now reversed, pares the remaining part of the edge upwards towards the junction of the fissure, where puckered up it remains until the other or right side is denuded in like manner. If possible the whole of the edge should be removed in one continuous piece from side to side, in order to insure the certainty that not the least particle of mucous membrane is left, otherwise perfect union cannot possibly take place. Some surgeons use scissors to denude the edges, but with such an instrument the parts are more or less bruised.<sup>1</sup> A needle such as this (fig. 13), armed with a thread, is then

FIG. 13.

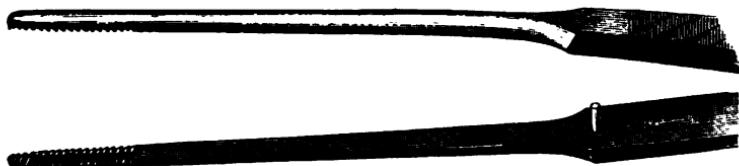


passed through the palate at about a quarter of an inch from the free edge. The thread is then grasped with either the

<sup>1</sup> Mettauer, 'American Journ. of Med. Science,' vol. xxi, 1837-38.

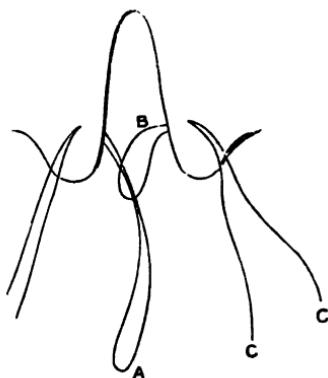
hook forceps or with a pair having serrated blades (fig. 14),

FIG. 14.



and the needle withdrawn. The needle is now re-threaded (or another may be used), and is to be passed through the opposite side exactly on the same level. If now the end **A** (fig. 15) be passed through the loop **B**, and traction made at

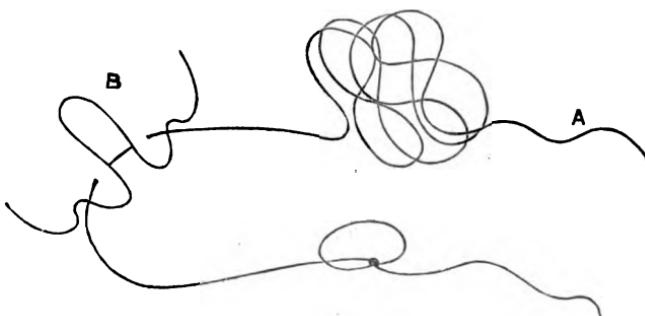
FIG. 15.



**C**, the end **A** will be brought through the opposite side of the fissure. It now only remains to pull through one side of the thread, when the appearance represented at **B** (fig. 16) is shown. When sufficient threads, say three or four, have been introduced, the next step is to approximate the edges. A slip knot is perhaps the best; and before putting the end into the noose, it is well for the surgeon to take the other end of the thread in a figure-of-8 form around his left forefinger and thumb, which manœuvre prevents the thread from getting entangled, and then it runs as easily as possible (fig. 16). Coloured threads may or may not be used. It is, I think, a good plan as the

operator proceeds to tie the ends of each succeeding thread; and supposing four sutures are employed, the practice I adopt is to

FIG. 16.



give the first thread, or that nearest the hard palate, to an assistant, who holds it at the centre of the forehead; the second is held *over* the patient's ears, the third *under* the patient's ears, and the fourth at the sides of the neck. Simple as this proceeding may appear, it saves confusion to a marked extent, for when the time arrives for drawing together the sutures, there is no difficulty whatever in selecting the corresponding ends. As a rule, I secure the stitches from above downwards. The operation is completed by either dividing the muscles, according to Sir W. Fergusson's plan, before the sutures are closed, if this has not already been done, or by taking the tension off the stitches by making a vertical incision, as Dieffenbach did, about a quarter of an inch in length on each side of the fissure. When necessary the anterior and posterior pillars of the fauces, with some fibres of the palato-glossus and palato-pharyngeus, may be divided. The accompanying woodcut (figs. 17, 18) show the incisions referred to.

Respecting the operation a few practical points may not be out of place. There is often some difficulty in grasping the thread when passed through by the needle, but it may be easily secured if the needle be thrust freely and somewhat roughly through and slightly withdrawn at once; but this must be done immediately and before the thread gets saturated with moisture. A slight loop in the ligature is thus formed, which

can be readily secured in the grasp of the forceps. Various instruments have been devised to catch the thread, but they are

FIG. 17.

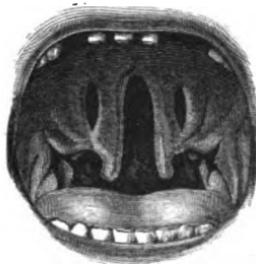


FIG. 18.



unnecessary, as a pair of forceps such as those already referred to answer the purpose perfectly. It is important, too, not to draw the stitches together too tightly, for there is generally a little swelling after the operation, and allowance must be made for this, but in referring to this point it must be distinctly understood that the edges must be applied with the most perfect precision, otherwise union cannot be expected. It is desirable, also, to place the knots so that they shall not lie exactly over the wound. In order to increase the breadth of the raw surface I have seen Sir William Fergusson take a curved knife and run it along the cut surface. This he thinks gives a greater probability of union.<sup>1</sup>

As to the length of time the stitches should remain is a point on which there is much difference of opinion. Sir W. Fergusson advises their removal about the third or fourth day; but then he was guided by circumstances, and has left them even to the eleventh day. I myself leave them to work their way out, unless they appear to cause irritation, when they ought to be taken away immediately. A remarkable case bearing on this question was under my care in February, 1876:—The patient was a boy aged 14; the stitches were left for one week, when a blush appeared all over the palate, and I was fearful that the parts might burst open. I removed the sutures, and on the following day all the inflamed appearance was gone, and the fissure united most perfectly.

<sup>1</sup> 'Med.-Chir. Trans.,' vol. xxviii, 1845.

As to the patient being confined to bed, I am of decided opinion that this is very necessary for three or four days. At all events, if he is not in bed he ought to be under the strictest supervision. Certainly in hospital practice this point is of great consequence, for the patient evading the nurse is apt to subject himself to draughts and to vicissitudes of temperature. Of this I had an example four years ago at the hospital.

The importance of absolute quiet, with perfect suspension of speech, is, I venture to think, somewhat overrated. Roux would not even allow the patient to swallow his saliva. It is as well that the patient should not speak above a whisper, and he should be provided with a slate and pencil to communicate most of his wishes. The sound advice given by Sir Philip Crampton, and by M. Ehrmann also, of not starving the patient should be rigidly carried out. All surgeons with any experience of staphyloraphy know that the operation is an exhausting one, sometimes there is considerable haemorrhage, and besides, the shock is great in certain patients, and is really severe in very young patients. There is in some cases considerable nausea and retching after the operation, hence it may be necessary to administer enemata of beef tea and other nutriment.

In reference to the disastrous effects of retching after the operation, I may refer to the case of a patient, a little girl, aged nine, sent to me by Mr. Wearne, and upon whom I operated. Two days after, she vomited two lumbrical worms, each about six inches in length, and the fissure broke open in consequence. Mason Warren attributed one of his failures to the sponges being filled with sand.<sup>1</sup>

I think the chief, and perhaps only real drawback to the use of chloroform is, that it is apt to be followed by nausea, retching, and vomiting. The patient swallows a good deal of blood, or rather perhaps the blood runs down into the stomach, which causes great uneasiness until that viscus is emptied.

In cases where there is oozing of blood the patient should be kept as quiet as possible, and be charged not to keep "hawking." I prefer that the mouth should be kept open, so as to allow the ingress of fresh and cool air rather than have recourse to the use of ice, for with the latter the chances of sloughing are increased by diminishing the blood supply.

<sup>1</sup> 'American Journ. of Med. Science,' April, 1848.

Steady pressure with the finger will arrest any haemorrhage, and I disapprove of the application of the perchloride of iron unless employed with great care and skill because it adds to the risk of sloughing.

As to the order in which the various stages of the operation may be performed there has been some difference of opinion. Thus, Roux divided the operation into three parts:—1st. He introduced the needles which he held in a sort of forceps, and passed them from behind forwards, using as ligatures four or five strands of thread well waxed; 2nd, he pared the edges of the fissure; and 3rd, he tightened the ligatures. He further detached the lips from the posterior border of the hard palate by a transverse incision of from four to six lines in length. Dieffenbach objected to this proceeding on the ground that, if the operation does not succeed, the soft palate is disqualified from another operation. Dieffenbach,<sup>1</sup> Mütter,<sup>2</sup> Velpeau and others, pared the edges before putting in the needle, and used leaden wire as sutures; Gräfe, Souchet, Jousselin, and Alcock, and more recently Sir W. Fergusson and Mr. Pollock, advocate the use of silk sutures, and this practice I most cordially endorse, after having tried silver, iron, and other materials, to bring the edges together. Mettauer recommended metallic sutures, and Sir Philip Crampton beads of metal. Iron, platinum, and silver wire have their supporters, but whichever is used it should be pliable. The wire such as florists employ is a very good material. Mr. Brooke used glass beads, and Mr. T. Smith prefers horsehair. Dr. Mason Warren and Professor Smith employed the surgeon's knot, believing that the first turn being double there is less risk of the thread slipping.

To favour union Dieffenbach made a longitudinal incision at four lines external to and on each side of the fissure. He says, "The side incisions are furthermore of particular importance. Only when the sides of the soft palate are pierced through is the operation, with anything or any way secure, and while without them we can only hope to close small openings in the palate, with them we are able to cure the largest, because by reason of the wide openings of the side incisions nature

<sup>1</sup> 'Lancet,' 1885, vol. i, p. 694.

<sup>2</sup> 'Brit. and For. Med. Rev.,' vol. xix, 1845, p. 412.

is forced to a regeneration by filling them up with granulations, so that the palate gains what it was deficient in breadth."

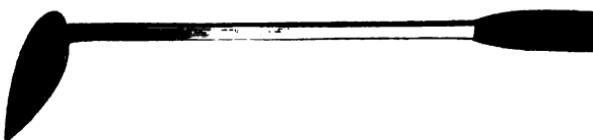
I have purposely left the question of the division of muscles in order that I might do full justice to the suggestions made by Sir W. Fergusson, Mr. George Pollock, and others. Moreover, inasmuch as chloroform, or some other anaesthetic, is now so uniformly employed, it seems a question whether the division of muscles as a primary step is so very important, and whether it may not be deferred until the last, when the parts may be released by dividing the sides of the soft palate. I now almost invariably adopt the latter practice with regard to the divisions of the muscles so as to arrest their action. Much has been done by Dieffenbach, Roux, Sedillot, Pancoast, Mütter, Mason Warren, Botrel, Avery, Pollock, and others; but it is I believe incontestable that to Sir W. Fergusson is due the credit of having, as Velpeau puts it, "*methodically applied myotomy to staphyloraphy.*" The all-important point in the operation is to insure temporary immobility of the parts; and Sir W. Fergusson, in his excellent paper published in the twenty-eighth volume of the 'Medico-Chirurgical Transactions,' 1845, placed his operation on a strictly anatomical and physiological basis; and proposed, "as an important accessory to the operation of staphyloraphy, that the surgeon should, on strictly scientific grounds, and in accordance with the modern principles of myotomy, so conduct his incisions as to destroy all motory power in the soft palate for the time being, and thus permit that repose of the stretched velum which is so essential to a happy result; in other words (says Sir William), I advise the division of the levator palati, the palato-pharyngeus, and the palato-glossus muscles. The first of these steps I deem of the greatest importance, the second scarcely less so, and the third may be effected or not as circumstances seem to demand." That Sir William Fergusson's views may not be misunderstood it will be best to give them in his own words:<sup>1</sup>

"Previous to paring the edges of the cleft, a knife such as this (fig. 19) is passed through the fissure, so that its point can be laid on the tissues immediately above the soft velum, midway between its attachment to the bones and the posterior margin, and about halfway between the velum and the lower

<sup>1</sup> 'A System of Practical Surgery,' 5th edit., p. 526.

end of the Eustachian tube; the point is then thrust deep, and carried half an inch or more backwards and forwards, so as to

FIG. 19.



cut the levator palati; next the uvula is seized with a pair of long hook-beaked forceps, and drawn forwards, so as to put the posterior pillar of the fauces on the stretch, which is then snipped across with long curved scissors, about half an inch behind the tonsil, by which cut the principal part of the palato-pharyngeus muscle will be divided; then, if it seem desirable, the anterior pillar of the fauces is touched with the scissors, so as to make the section of the palato-glossus, a proceeding which I scarcely deem requisite."

With regard to the actions of the muscles in cases of cleft palate, Sir William gives these as his conclusions:

"1st. That the flaps are slightly drawn upwards and to the sides, when the levator palati contracts.

"2nd. That when the levator palati and palato-pharyngeus act strongly and together, the flaps are so forcibly drawn from the mesial gap, that they can scarcely be distinguished from the sides of the pharynx.

"3rd. That the flaps are forced together and the edges come into contact, when the superior constrictor muscle contracts during the act of deglutition.

"4th. That the circumflexus palati possesses but a feeble power over the flaps.

"5th. That the fibres of the palato-glossus are very imperfectly developed in the specimen in his possession."<sup>1</sup>

It is well to remember that, from some constitutional cause, and quite independently of muscular action, the parts may break open. On this point Avery<sup>2</sup> remarks, "It should be particularly noted that this separation does not always take

<sup>1</sup> 'Brit. and For. Med. Rev.', April, 1845, No. 38, p. 415.

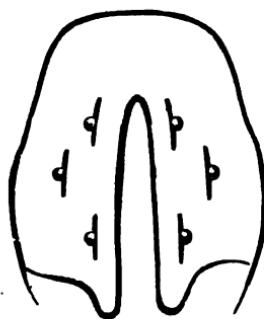
<sup>2</sup> 'Lancet,' 1852, vol. ii, p. 31.

place because the parts are *torn asunder*, but because they have failed to unite."

Pancoast, referring to his method,<sup>1</sup> says, "When the knots are prepared for tying, but before they are finally secured, Wenzel's cataract knife is passed from before backwards through the attached sides of the palate, thus, to enable the two halves of the velum to come together in the middle line, as well as to divide the insertion of the palate merely so as to prevent their straining the sutured edges of the palate asunder."

Warren divided the anterior and posterior pillars, and M. Sedillot, alluding to his own practice, says,<sup>2</sup> "My incisions pass through the entire thickness of the velum palati, and are a continuation of the lateral divisions of Dieffenbach, Pancoast, Liston, and Warren, of the anterior and posterior pillar of the fauces. Mettauer released the parts by a number of small lateral incisions (fig. 20). (This woodcut is copied from his paper.) Dr. Smyly<sup>3</sup> recommended the division of the muscles

FIG. 20.



something after Sir William Fergusson's method, only that he put the knife along the floor of the nose.

Mr. Callender, in order to obviate the difficulty arising from haemorrhage, says,<sup>4</sup> "I divided the levator palati on either side, and five days after I passed four wires through the side of the fissure, and the palate being held forward and steadied

<sup>1</sup> 'American Journ. of Med. Science,' vol. xxxii, 1843.

<sup>2</sup> 'Med. Times,' 1850, p. 375.

<sup>3</sup> 'Med. Times,' June 7th, 1862.

<sup>4</sup> 'Clin. Soc. Trans.,' vol. i, p. 173.

by means of the wires, I proceeded to pare the margins, and subsequently brought them together by twisting the wire."

Mr. Pollock arrests the action of the muscles in the following manner:—"First, he says, a suture is passed through one section of the soft palate at the root of the uvula, the ends secured together by a knot, and held outside the mouth. A second suture is then passed through the opposite side at a corresponding point. One of the sutures, now firmly holding one half of the soft palate, is drawn gently forwards and to its opposite side, so that the section of the palate is well stretched towards the median line. A thin, narrow, sharp-pointed knife, fixed in a long handle, is then introduced into the palate, close to the hamular process, a little in front and to its inner side. This process can be distinctly felt in the substance of the soft palate, internal and a very little posterior to the last molar tooth. Running the knife upwards and backwards, and somewhat inwards, the point may at last be seen in the gap, having passed through the entire thickness of the soft palate, and having cut, if not wholly, at any rate partially, through the tendon of the tensor palati: the knife should now lie above most of the fibres of the levator. If the handle of the knife be next raised the point becomes depressed; and if the blade be drawn forward, while it is at the same time made to cut downwards, it travels through a considerable section of a circle on the posterior surface of the palate, and insures the division of the greater portion of the levator palati. As the knife-blade travels downwards, the tension of the palate gives way, and often the division of the muscle is felt to be suddenly effected; the ligature being no longer pulled upon by it, though previous to division it will be felt sensibly and spasmodically contracting. As the knife is withdrawn through the wound, the division of the levator muscle should be thoroughly effected. The wound in the front of the palate need be no more than the width of the knife; whereas the wound behind is necessarily much longer, for the fibres of the levator have there to be divided by the sweep of the knife. Provided the muscle be effectually divided, as soon as the knife is withdrawn it will be found that all voluntary and involuntary movements of the palate have ceased; it has become pendulous and flaccid; pulling on it now should produce no spasmodic

contraction of its fibres. Should any resistance still be observed, the knife must be again introduced through the anterior wound, and the fibres a little more freely cut in a downward direction.”<sup>1</sup>

## 2. CLOSURE OF THE HARD PALATE.

- a. By Uraniscoplasty.
- b. By Osteoplasty.

### a. *Uraniscoplasty.*

The idea of closing the hard palate is said to be due to Dr. Mason Warren, but, as already stated, M. Krimer effected this object in 1824, and in the following manner:—“He made a semi-elliptical incision comprising the whole thickness of the palate on each side, two or three lines from the fissure; he then dissected off the two flaps and reversed them (Procédé par renversement, as the French surgeons call it) from without inwards towards the middle line, and then united them with a suture.”<sup>2</sup> M. Beaufils made a *single* flap, and twisted it upon itself to fill the aperture.

“The method of proceeding originally proposed by myself (says Mason Warren) in 1843 was as follows:—First, when the bones composing the arch of the palate were divided, to dissect off the mucous membrane covering them on each side as far as the alveolar processes if necessary, stretching it across the fissure, and confining it in this situation by sutures; the flaps, it must be understood, being made continuous with the fissured halves of the soft palate. Second, in the above cases, and in fact in all where the lateral halves of the soft palate are too small to be easily brought in contact, as generally happens where the bones are involved, to cut away the posterior pillars of the palate with strong curved scissors, and continue the dissection ‘behind the soft palate until the latter yields and

<sup>1</sup> ‘Holmes’s System of Surgery,’ vol. iv, article “Diseases of Mouth.”

<sup>2</sup> ‘Dictionnaire de Médecine et de Chirurgie Pratiques,’ vol. xv, 1836, p. 19.

allows itself to be drawn across the chasm, which, by the above proceeding, will be found practicable, even in those fissures which at first do not seem to offer the slightest hope for a successful operation."

In cases that are complicated with harelip it is, I believe, the best plan to operate as early as possible on the lip, for in this way the cleft in the palate becomes in a short time very much diminished in size, and much more amenable to treatment by operation. Further, I am convinced of the advantages of closing the lip, whether there be a cleft in the palate or not, as soon after birth as possible, for whilst the little patients seem to fade away before surgical interference, they thrive immediately and gain flesh rapidly after the operation. I have now under my observation several cases to prove this point.

Passavant, of Frankfort, relates the case of a child whose harelip was closed at the age of nine weeks, and a year after the palate was found to be so approximated without further operation that it presented a mere fissure (*raphé*).<sup>1</sup> Duplay and Rouge express their opinion thus:—"That in bad cases in which life is involved the lip should be dealt with as soon as possible after birth, and not to close the hard palate until about the end of the first year, and to reserve the operation on the soft palate, say until six or seven years of age."

Prolonged compression on the two maxillæ has been strongly recommended, and is no doubt of service in certain cases. Dupuytren, Jourdain, Levret, have much confidence in this practice, and MM. Autenrich and Mannoir employed an instrument which is very like that known to English surgeons as Hainsby's compressor.

Langenbeck, in his 'Archives de Clinique Chirurgicale,' 1861, t. ii, p. 280, states that in 1845 he tried what he terms "*the bony suture*" in a child aged three months, who had cleft palate and double harelip, with the intermaxillary bones quite isolated. He says, "I turned the piece back after cutting through the cartilage, and I fastened it on each side to the alveolar border with a leaden thread, which I twisted in the mouth. I then operated on the harelip: the case succeeded very well. On the sixth day after, the lead sutures were removed; suppuration, however, took place in their track, and at length three

<sup>1</sup> 'Archiv. f. Klin. Chirurgie,' t. v, p. 52.

teeth came away." He candidly admits, however, "that as the sutures had traversed the dental follicles, an accident which cannot be provided for, he had not thought fit to repeat the operation."

On the other hand, some authorities<sup>1</sup> recommend that the palate should be closed before the lip is interfered with, on account of the increased accessibility of the parts.

Assuming the case to be one of fissure extending through both the hard and soft palate, the question has arisen whether the soft part should be closed first, or whether the hard part should take precedence—the cure being completed in two operations—or whether the whole of the fissure should be closed at one operation. Here again there is difference of opinion. Thus Sedillot and Passavant recommend staphyloraphy first, then uraniscoplasty. Langenbeck, Erhmann, Rouge, and Pollock, on the other hand, advocate closing the hard palate first; indeed Mr. Pollock,<sup>2</sup> writing in 1856, says, "The attempt to unite the hard and soft palate at once is an extremely injudicious proceeding, and will most likely end in failure." He further prefers to commence with the anterior part when the fissure extends in the maxillary bones. I have, however, in several instances closed the entire fissure at once with the best results, and have found that, even if the soft part breaks open, the hard palate as a rule unites very favorably. M. Rouge found that out of twenty-eight cases he had seen of uranisco-staphyloraphy done at one operation, only ten were completely closed at once. Billroth had only three successes out of eight, and Langenbeck one only out of four.

Pancoast<sup>3</sup> thus describes his method of staphyloplasty:—It consists, he says, in a partial division of the two sides of the cleft near their bony connection, so as to admit of the middle strips being readily brought together, or by the raising of flaps from the side or the roof of the mouth, which are to be turned over and fastened by suture in the middle line.

Langenbeck<sup>4</sup> claims the right of priority for this kind of operation, and states that he was the first who *completely* closed

<sup>1</sup> Rouge, *op. cit.*

<sup>2</sup> 'Med. Chir. Soc. Tr.', vol. *xxxix*, 1856.

<sup>3</sup> 'American Journ. of Med. Science,' vol. *xxii*, 1843, n. s. 6.

<sup>4</sup> 'Med. Times,' Jan. 11, 1862.

the hard palate by *déplacement* or *glissement*. Duplay, however, remarks that although Dieffenbach, Avery, Baizeau, and Langenbeck claim the priority, yet Baizeau was the first to put the plan clearly before the profession. Langenbeck appeared to be the first to insist on including the periosteum with the mucous membrane. According to Rouge this was first done by Langenbeck in 1860. Yet, referring to a case published by Mr. Avery,<sup>1</sup> the writer says:—"The most interesting portion of the operation was the difficult task of detaching the tough tissues adherent to the hard palate and lined with mucous membrane." And Mr. Pollock comes to the rescue of his countryman when he says,<sup>2</sup> "I should not be doing justice to the memory of the late Mr. Avery if I omitted to mention that he was the first surgeon in this country to close entirely a complete cleft of the palate, and that the operation which Professor Langenbeck proposed, and to which he gave the name of 'the operation of muco-periosteal flaps,' appears to be identical with the method of operating introduced by Mr. Avery."<sup>3</sup> He further states that in 1848 Mr. Avery first succeeded in closing clefts of the hard palate by operation, and that in 1853 Messrs. Weiss made raspatories for the performance of the operation. I need not add that the separation of the mucous membrane without including some of the periosteum is well nigh an anatomical impossibility because the two structures are so intimately connected.

Pollock says the incision for closing the hard palate should be made close to and parallel with the alveolar ridge, and extend from a point opposite to the last molar forwards to the canine tooth. Writing in 1856, Mr. Pollock says that he separated the soft parts in a direction "from the fissure to the alveolus," and then made a cut along the alveolar border. This takes off all tension. But more recently (1870) he remarks:—"I have adopted the plan of commencing from the incisors and proceeding inwards, terminating when the edge of the gap has been attained." "The flap," he adds, "should consist of all the

<sup>1</sup> 'Lancet,' vol. ii, 1852.

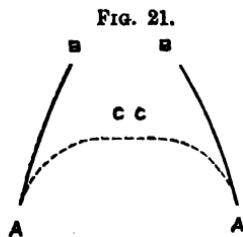
In Holmes's 'Surgery,' p. 436, footnote.

See Prof. Langenbeck's treatise entitled "Weitere Erfahrungen im Gebiete der Uranoplastik mittelst Ablösung des Mucös-periostalen Gaumenüberzuges," Berlin, 1863.

soft tissues covering the bone—mucous membrane, areolar tissue, &c." It is highly probable that Dieffenbach performed a very similar operation many years previously, for he states:—"If the opening in the hard palate be large, and the edges covered with a thin skin, the borders are cut round within about a quarter or half an inch of the edge. The skin is pushed away from the bone with a scraper, and the opening fastened by a suture. The side wounds are filled up with charpie and treated as usual."

The great advantage that Langenbeck claimed for the separation of the periosteum was that the new palate is composed of bony substance. "The osseous formation," he remarks, "takes place about the third week after the operation. It is completed at the end of the fourth week, and afterwards attains considerable solidity."<sup>1</sup> He tried it with a needle, and believed that ossification had really taken place. Doubts, however, have been thrown on this point, for it was supposed that the toughness was due merely to cicatricial tissue, which is well known to be very unyielding. To prove this point M. Marmy tried some experiments to ascertain the results of operation on dogs' palates, and found that although union was exceedingly tough, and almost as hard as bone, yet there was no true osseous tissue formed. The nature of the material is, however, of little practical importance, and M. Ollier, the originator of the subperiosteal resections, puts the case in its proper light in saying:—"If there may be doubt as to ossification, all must admit that it forms a very resisting surface which has the strength and takes the place of bone."

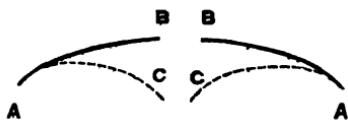
The success of the operation depends greatly on the extent of the arch of the palate, for if the part be of this shape (fig. 21)



<sup>1</sup> 'Archiv für Klinisch. Chir.', vol. v, 1er cahier, p. 3.

it is obvious that when the sides (A B) are detached they will fall together more readily (A C) than if the arch be formed thus

FIG. 22.



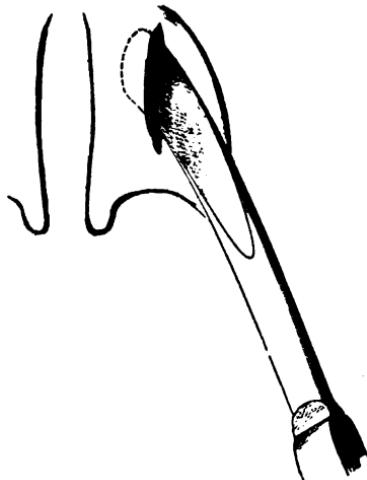
(A B, A C, fig. 22). Mr. Pollock remarks on this point, that  
"The more complete the cleft the nearer the perpendicular are

FIG. 23.



the sides of the palate, and consequently, when the soft tissues are detached from the bone, the flaps formed fall inwards, and very readily meet in the median line."

FIG. 24.



With reference to these different operations of uraniscoplasty

I am decidedly in favour of the so-called Langenbeck plan. I feel sure that a much thicker and stronger flap can be taken away if the respiratory be introduced near the alveolar border of each side and made to work its way towards the fissure. In 1865 I had under my observation a case illustrating the advantage of that procedure. I had operated once before on the same patient by separating the soft tissues from the hard palate, dissecting it off with a rectangular knife (such as that depicted in fig. 23) *from the fissure towards the alveolus on each side*. The operation failed signally. In about a month I operated by the so-called Langenbeck method, using an instrument of this kind (fig. 23) and applying it as here depicted (fig. 24), and obtained a strong, thick flap from either side, and the success was all I could possibly expect.<sup>1</sup>

#### *b. Osteoplasty.*

With regard to osteoplasty there is little doubt that Dieffenbach was the first to suggest this practice. In 1826 that surgeon detached on each side with a saw or scissors a straight portion of the hard palate to free the osseous portions, and to make them approach the middle line. The parts were kept together by little wedges of wood and a metal suture.<sup>2</sup> Here are Dieffenbach's own words respecting the operation:<sup>3</sup>— "The edge of each palate bone is pierced with a strong, straight, three-cornered punch, and a thick soft silver wire put through the opening, the ends of which are twisted together. The mucous membrane is divided near the place where the palate bone joins the alveolar processes; a thin, smooth, concave chisel is then put to the bone, and it is cut through on both sides. The wires are then twisted again till the edges of the bony cleft approach each other a little, or altogether. The first alone can generally be done. The ends of the wire are then cut off. The effect of the closer approximation of the edges of the cleft

<sup>1</sup> 'Med. Times and Gazette,' January 28, 1865, p. 87.

<sup>2</sup> Rouge, op. cit., p. 15.

<sup>3</sup> 'Die Operative Chirurgie,' von Johann Friedrich Dieffenbach, Erster Band, 1845, p. 856.

in the bone is immediately perceptible in the soft palate. The side slits in the bone, which are at first filled up with lint, close themselves by means of granulations, according to the same process. When the space in the bone is either closed or diminished so much that the cleft in the soft part is considerably lessened, the sawing of the palate may then be undertaken according to the direction already given, and side incisions made in the soft palate before the sutures are put in. The operation may be continued from time to time until the cleft is removed."

In a very interesting and practical paper by Sir W. Fergusson, entitled "Observations on Harelip and Cleft Palate,"<sup>1</sup> this distinguished surgeon refers to an operation which he believed to be novel, but which is in reality very similar to that proposed by Dieffenbach. Sir William's results appear to be much more encouraging than those of the continental surgeons; thus, Rouge speaks of five cases in which this method of procedure was adopted, and all of which failed from necrosis; again, from 1849 to 1856 Langenbeck operated on three cases with unsuccessful results.

Adopting Sir William Fergusson's plan, I found, in the first two or three cases on which I operated, that there was some exfoliation of bone, and I venture to think that the necrosis depended on the somewhat rough way in which the bone was divided. By simply pushing the instrument through the bone it is apt to splinter, and in order to obviate this I have since adopted a very simple method, which I brought before the notice of the profession in 1874.<sup>2</sup> It consists in boring holes with an ordinary bradawl (fig. 25) on each side straight

FIG. 25.



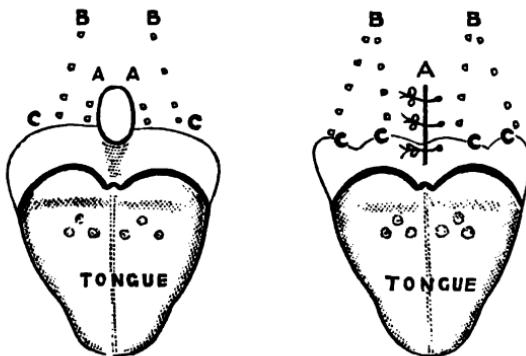
through the hard palate, exactly in the line in which the chisel

<sup>1</sup> 'Brit. Med. Journ.', April, 4th, 1874.

<sup>2</sup> 'Lancet,' October 24th, 1874, p. 578.

is to be applied (fig. 26, B c). The least pressure with such an

FIG. 26.



instrument as this (fig. 27), which is really nothing more than an ordinary screwdriver with a sharp edge, will at once divide the bone without splintering. The proceeding is extremely simple, and may not be inaptly compared to the perforated edges of postage and other stamps.

FIG. 27.



The sutures may be applied with a pointed needle, as already described, or one with a blunt point, such as this (fig. 28) may be employed.

FIG. 28.



In closing the hard palate by this method there is often a good deal of haemorrhage. Hence the operation should be performed as speedily as possible, but without undue haste. Either in the so-called Langenbeck's operation of uranisco-

plasty, or in Dieffenbach's osteoplasty, the bleeding may be instantly arrested during the passage of the sutures by stuffing the sides with a piece of lint of suitable size; I am sure from experience that this is a most useful expedient, and the lint may be allowed to remain after the operation or another piece of proper size introduced in order to give support to the sides. As already stated, Dieffenbach in 1826 used little wedges of wood for this purpose, and MM. Sedillot and Gustave Simon inserted small pads of cotton wool in the incisions.

If osteoplasty be performed without chloroform it does not seem to be attended with so much suffering as uraniscoplasty, for in the latter the separation of the soft from the hard palate is a somewhat painful proceeding.

In selecting between the two operations above described for closing a fissure of the hard palate the surgeon must consider, first, the shape of the palate, and, secondly, the amount and thickness of the soft tissue covering it. My personal experience of Dieffenbach's operation of osteoplasty is that even with the greatest care exfoliation of bone to a greater or less extent not infrequently takes place, as already stated. The operation has not been received with favour by Continental surgeons, and whilst I advocate its performance in suitable cases, I am nevertheless satisfied, from the large number of patients under my observation, that uraniscoplasty—the so-called Langenbeck method—especially on account of the less risk of exfoliation if efficiently performed, is generally followed by equally successful results.

I may mention that in such cases as those in which the bone is adherent to one side of the palate a slight modification of the operation may be required, and the surgeon must be guided by circumstances. Thus, in the case depicted in fig. 6 I detached one side by a bridge-like flap including the bone, and denuded the opposite surface by the muco-periosteal operation. The case did very well.

The operation for closing a congenital fissure of the hard and soft palate is certainly not attended with any special danger to life. The haemorrhage, it is true, will frequently exhaust the patient to a considerable extent; but in all the experience that I have by the kind friendship of Sir W. Fergusson derived I have never seen or heard of one single case of death as the

immediate result of the operation. There are, however, such cases reported, but only in very young children; thus, besides those to which I have already alluded Dr. Ehrmann mentions one instance of death from haemorrhage in a child seven and a half months old,<sup>1</sup> and also refers to four fatal cases in infants in whom the operation on the hard and soft palate had been attempted, one of four days old, one of five days, and two of two months old.<sup>2</sup> In a case of M. Gustave Simon's the flap sloughed and the child died of septicaemia, then a patient of M. Berard's, as well as one of Maissonneuve's, died of erysipelas of the face.

#### ON THE IMPROVEMENT OF THE VOICE AFTER THE OPERATION.

The chief object of the operation, whatever plan be adopted, is obviously to improve the voice of the patient, and I have no hesitation in saying that in many instances the voice is very materially altered for the better. It is too much to expect that the sufferer should speak as fluently as his neighbours whose palates are normally developed. Langenbeck thought that the nasal twang in cleft palate was due to want of nerve supply, but there is reason to suppose, as Passavant and Gustave Simon do, that it is attributable to the shortening of the palate.

As to the improvement in the voice after the operation, Mason Warren<sup>3</sup> refers to the case of a young man who spoke at a public meeting about two years after the operation, and it was difficult to discover the least imperfection of his speech, although previously he had been excluded from society. Again, in a curious case of Mr. Wardrop's<sup>4</sup> it is stated that "the patient, a girl, aged twenty-one, who was passionately fond of music, was able to sing with considerable execution." And another remarkable case is recorded<sup>4</sup> in which a patient, a Frenchman, could express himself equally well in French and in English, but his voice had a nasal twang when he spoke French, and was almost normal when he spoke English.

<sup>1</sup> 'Lancet,' August 20th, 1870, p. 259.

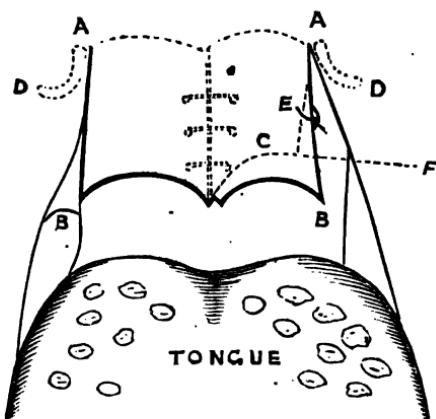
<sup>2</sup> 'American Journ. of Med. Science,' April, 1848.

<sup>3</sup> 'Lancet,' vol. xii, p. 350.

<sup>4</sup> 'Dictionnaire de Médecine et de Chirurgie Pratiques,' vol. xv, 1836.

I venture to think the voice may be still further improved by the simple operation I suggested in 1869, an account of which was published in that year.<sup>1</sup> Referring to this point I may be permitted to remind the reader that a cleft in the palate is not a mere rent or slit in the parts, but an actual deficiency or want of tissue. Hence, however well satisfied the surgeon may be with his work, the soft palate still remains as a tight curtain stretched across between the mouth and posterior nares. The result is that, in speaking, the air instead of passing into the mouth gains ready access to the nostrils, and thus the peculiar nasal twang is occasioned. In order to obviate this I release the soft palate in the following manner. The operation may be performed at any time after the complete closure of the soft palate, say a month or more:—A small curved spatula is first placed behind the soft palate; it keeps the part steady and also serves as a *point d'appui*. A sharp-pointed knife is then introduced from before backwards at A (fig. 29), in about the position of the inner edge of the

FIG. 29.



hamular process in the normal palate (D), and the soft palate is cut completely through from above downwards from A to B. The same thing is repeated on the other side, and the operation is then concluded. In the first few cases on which I operated

<sup>1</sup> 'Lancet,' vol. ii, 1869, p. 198.

I hemmed the mucous membrane, back and front, as indicated in the diagram *x*, but I have long since abandoned this practice as unnecessary, for when the parts unite they do so at the V-shaped angle where these are in immediate contact (dotted line *r*). The operation is very simple and may be repeated as often as necessary, is perfectly free from danger, and almost painless. The rationale of the proceeding is easily explained. The palate becomes converted into a huge uvula, so to speak. It is shortened and puckered up, the point *s* being drawn up to *c*, so that if it does not actually touch the back of the pharynx it approaches it so nearly as to divert the current of air to a considerable extent from the nose into the mouth, and thus greatly diminishes the disagreeable guttural voice that is more or less observable in all patients who are the subjects of this distressing deformity.

Still, the improvement depends to a great extent on the care and intelligence of the patient. I have met with some cases in which the voice has been almost perfect.

#### ACQUIRED OR ACCIDENTAL APERTURES IN THE PALATE.

These may result from injuries, or after the partial removal of the upper jaw or from antral tumours invading the mouth, but complete fissure from disease is rarely if ever seen. M. Jobert (de Lamballe) reports a case of perforation after an attack of measles. Necrosis and exfoliation of the palatine plates of the palate or superior maxillary bones is attributed by high authorities and by almost universal consent to a syphilitic taint. That such cases are necessarily syphilitic is in my opinion open to question. Necrosis may, of course, occur in a person who has had his constitution affected through the true infecting sore, but I have irresistible evidence to show that in most of the cases that have been under my observation there has not been a particle of history of that disease. I now take the opportunity of repeating my own experience (and it is my individual experience only) that with nearly twenty years' hospital and other practice I have never yet met with a single patient who has been under my care, or whose case I have had the opportunity of watching, *from the first* with

an infecting sore (followed by psoriasis of the palms of the hands accompanied with sore throat, condylomata, &c., these symptoms being, I presume, typical of constitutional infection), who has ever returned to me, or has been brought to me, with exfoliation of the bony palate or of the nasal bones. With such facts before me I must hesitate before I accept the broadcast belief that syphilis is in such instances the *fons et origo mali*.

I have invariably noticed that ulcerations of the palate complicated with exfoliation of bone occur in pale, ill-nourished, and cachectic people who, if there be ulcerations on other parts of the body, say the face or in the neighbourhood of the joints, are soon, I may almost say instantly, benefited by five-grain doses of iodide of potassium, with some preparation of iron administered thrice a day. It might be argued that because the administration of iodide of potassium is curative, that this fact is proof positive that the case is syphilitic; but such an argument is untenable. The truth is that the drug is useful in all diseases in which iodine is indicated. It certainly has a marvellous effect on such ulcerations.

Apertures in the hard palate are admirably suited for mechanical appliances, such as an obturator, but the instrument should be fitted with the greatest accuracy lest the pressure on the lowly vitalized part should induce further ulceration. Mr. W. D. Napier, Mr. Hamilton Cartwright, and other surgeons practising dental surgery, have drawn attention to this point. It often happens that in these cases patients stuff the opening with some soft substance, such as lint, sponge, wax, cork, crumb of bread, papier maché, &c. This is, however, a most pernicious practice, for simple and efficient as are the means employed to improve the voice, yet the improvement is effected at the expense of the opening, the continued pressure causing a steady increase in the size of the aperture.

Various obturators have been suggested, the first instrument of the kind probably being one used by Petronius in 1565. At the present day there seems to be no end to the ingenuity displayed in making such apparatus. Ambrose Paré in his marvellous work published in 1649 (English edition) gives two woodcuts, one in which there is a plate of silver to which is attached a piece of sponge, by the swelling of which

the plate is held in the aperture, and another on whose upper side there is "a button, which may be turned when it is put into the place, with an instrument like a small raven's bill."<sup>1</sup> In the 'Lancet'<sup>2</sup> will be found illustrations of Weiss's instruments. Some appliances are fastened by rings, some by bolts, and some are fixed to the teeth.

The impairment of the voice depends greatly on the position of the perforation; thus the voice is perceptibly altered if there be even the smallest hole in the bony palate, and to a less extent if an aperture be in the soft palate. I may here refer to the singular fact that the voice is, in many instances, comparatively scarcely impaired, even if there be extensive adhesions of the soft palate to the back of the pharynx. I have had under my observation at the hospital and elsewhere numerous cases illustrating these points.

Some strangely heroic operations have been suggested and even performed for closing such apertures, which, however, scarcely merit imitation; thus, in 1836 Regnoli closed a hole in the palate after resection of the superior maxilla by taking a piece of skin from the upper lip, and Blasius took a flap of skin from the forehead in a case where there was no nose. Again, at a medical meeting at Leipzig Professor Thiersch showed a patient in whom uraniscoplasty had been performed for acquired defect of hard palate where obturators could not be borne. The cleft was closed by transplantation of the skin of the cheek. The flap healed perfectly. There were still some small fistulæ between the nose and mouth. The epidermis bristling with hairs was seen in the cavity of the mouth.<sup>3</sup>

Respecting small holes in the soft palate which remain either after the operation of staphyloraphy or after ulceration, the application of either nitric acid or lunar caustic will in most cases effect their closure. Dieffenbach believes that the best application is the tincture of cantharides.

Any attempt at closing an aperture in the soft palate acquired by constitutional disease by operation will almost invariably fail. I have had the privilege of assisting Sir W. Fergusson in

<sup>1</sup> Lib. xxiii, p. 579.

<sup>2</sup> 'Lancet,' 1827, vol. iii, p. 325.

<sup>3</sup> 'Med. Times and Gaz.,' January 16th, 1869, p. 75.

many operations of this kind, and he was invariably of the same opinion. Indeed, Sir William's more recent experience may be summed up in the same words he used at a clinical lecture delivered in 1852:—“I must tell you, gentlemen, that in cases of this description, where there is an opening in the soft palate produced by disease, there is very little chance of doing good by an operation. I have tried on various occasions to close openings of this nature, but cannot flatter myself with being successful.” I must confess that my personal experience tends to support this statement.

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As frequent reference has been made in the above paper to the name of my esteemed friend the late Sir William Fergusson, I think it right to state that the article was completed before his death, and that it is now produced exactly as it was originally written.

<sup>1</sup> ‘Med. Times,’ vol. xxv, May 1.



ON THE  
WORKING OF THE ADULTERATION ACT.

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By ALBERT J. BERNAYS.

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SINCE my last report printed in our 'Reports' for 1875 the amended Act on the sale of food and drugs has come into full work, and we can judge of its merits and failings.

The experience gained as to the nature of the alcoholic drinks generally sold by the licensed victuallers and grocers has somewhat disabused the public mind with reference to any gross adulteration, but it is at present quite insufficient to enable one to speak with certainty as to their quality.

One of the greatest abuses is connected with the VARYING QUANTITY of ALCOHOL contained in fermented liquors. This uncertainty is a fruitful cause of drunkenness and demoralisation, and this portion of the chapter on LICENSING is certainly capable of further legislative treatment.

A glass of gin in one place contains 50 per cent. of proof spirit; in another 76 per cent.

Aniseed, which is given to young children, contains no spirit at one public house; at two other houses respectively, it contained 82 and 40 per cent.! The differences amount to no poison and poison, and are such as should be rendered impossible. It is also a noteworthy fact that public houses celebrated for the immorality of their patrons are those in which the strongest alcoholic liquors are sold. Such being the case, the question

arises, What can be done to assist those who are morally too weak to help themselves?

I certainly agree with Mr. LOWE as to the licensing system, and have already, in the 1862 edition of my 'Science of Home Life,' expressed myself sufficiently strongly with regard to it. I there wrote of "the utter profligacy of the licensing system, the only cure for which would lie, as the author believes, in complete free trade." Now, I do not mean to say that the trade in liquor would not have to be placed under more stringent law, nor but that, at first, an increase of the mischief would follow from free trade. I would venture to recommend what the 'TIMES' of December last was obliging enough to insert,—that all spirits containing above 50 per cent. of proof spirit should, for each fraction between 50 and 60 and upwards, be designated by certain brands, and should be paid for at a higher rate. Thus, no spirits should be sold of less alcoholic strength than such as contained 50 per cent. That of 60 per cent. might be characterised as X; that of 70 per cent. as XX, &c. In this way something might be done for the protection of the public against themselves, and the criminal law should do the rest.

Another question which has not yet been settled by public analysts arises as to the QUALITY of the alcohol in wines and spirits. The more rectified the spirit, and the more it approaches to pure ethylic alcohol, the less injurious it will prove. But here is the difficulty! The new Act requires the sample to be divided into three parts. As soon as the contents of a bottle of wine shall have been divided into three parts, the quantity apportioned to the analyst is too small to give anything but a qualified opinion as to the purity or otherwise of the alcohol. Often a very cheap corn brandy is employed for fortifying the different brands of port and sherry; and, as this corn brandy is very incompletely rectified and contains alcohols in addition to ethylic alcohol, such wines are far less digestible and more injurious than the wholesome natural wines of France and Germany.

If people would only be satisfied with the light wines last mentioned, for which we owe a great debt of gratitude to Mr. GLADSTONE, many of the well-founded complaints about the drunkenness of our people would vanish.

But even our national drink, BEER, is found to vary in a most extraordinary manner with regard to proof spirit. It is not uncommon to meet with porters varying from 5.32 per cent. to 12.76, and this from public houses almost side by side. Can it be wondered at that a man, who frequents such houses for a draught, is sometimes overtaken when he has really no wrong intention? As far as the power of quenching thirst is concerned, the less of proof spirit the better; the thinnest table beer would be best; and, as far as the power of doing work is concerned, the more complete the absence of any alcohol the better! The chief excuse for beer is to be found in the quality of the water, and the greatest boon to the public would be an alcoholically weak beer.

COMMON SALT was once employed as an adulterant; it is now rarely met with. In most of the cases with which I am familiar salt is present in remarkably small quantities. Indeed, if beer were generally brewed with a hard water, it would keep better and require less proof-spirit. If I am asked whether hops are yet to be met with in beer, I can only say "occasionally."

With reference to the COLOURING MATTERS of red wines I have no experience of any adulteration, and I have reason to believe in the extravagance of the assertions put forward.

The most shamelessly adulterated articles at the present time are MILK and BUTTER. Leave the Act in abeyance in any parish for three months, and the deterioration of milk by water is as certain to grow into vigour as the sprouting of the trees in spring. The penalty put upon adulterated milk by some magistrates is so small, and this very subject of adulteration is by so many regarded as a joke, that the practice is sure to continue. And yet, when we think upon the quality of the water that may be admixed, and upon the importance of milk as an article of diet, the deterioration of milk is a great iniquity. The working of the Act is very difficult, as many milkmen at once admit that they are selling milk and water, and thus prevent an analysis from being made. It is a pity that the names of such should not be simply placarded, as that plan would undoubtedly act as a deterrent. And yet I should be sorry if the truth were not apprehended. Four

years ago the adulteration of milk was the rule ; now it is the exception.

One of the many difficulties connected with the present working of the Act lies in the removal of **MIXTURES** from the category of adulteration. A packet of so-called cocoa, coffee, or mustard has only to be labelled as a mixture, and there is then no further protection to the public as to whether cocoa, coffee, or mustard be or be not the leading constituent ! Under this heading of "mixtures" the old adulterations will creep in, unless the vestries are made alive to the necessity of examining such mixtures with reference to the purity of the articles used.

A curious case is now before one of the magistrates. Green peas have been introduced which owe their colour to about one third or more of a grain of a salt of copper in each pound. The public analyst (Mr. PIESSE) objects to the presence of copper, but scientific men have given evidence that such a minute dose would only act as a tonic. Now, considering the impossibility of keeping the amount of copper within any certain limit, and the probability that some of the peas might be far more largely poisoned than others, the copper should not be allowed. The 'Lancet' did good work in days of old in putting a stop to the colouring of pickles and preserved fruits, and it would indeed be a scandal if the old system were to come into vogue again on the plea of the smallness of the amount of contaminant.

Of the **CUMULATIVE EFFECTS** of minute quantities of **METALLIC CONTAMINATIONS** I may give the following illustration. A man was brought into one of our metropolitan hospitals afflicted with some of the worst symptoms of lead-paralysis. For some time the physician in attendance could not in any way account for the man's illness, as he was not connected with painters' or any other work in which lead was employed. At length it came out that the man in going to his daily work stopped every morning at the same public house, and had a draught of "dog's nose," a mixture of gin and ale. The gin having been in the pipes all night was just sufficiently poisoned by a trace of lead as in time, by its cumulative effects, to produce paralysis. Among other things it shows that the fittings of a public house are not unimpor-

tant, and that the mention of distinct traces of lead and copper are not to be disregarded.

Many MEDICINES examined are of first-rate quality. A curious instance to the contrary I have found in a sample of trisnitrate of bismuth. It had been so baked and was so hard and lumpy that it is extremely doubtful whether it would have been at all absorbed. The physical condition of a powder is of first importance, and it is still a question whether smaller amounts of medicines might not be employed if more attention were paid to keeping them in the state which they assume when first precipitated from a solution, and to the temperature of the body, which gives a greater solvent power than is possessed by liquids at the ordinary temperature of the air.



ON THE DIFFERENT FORMS  
OF  
HYDROCELE OF THE TUNICA VAGINALIS.

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By SAMUEL OSBORN, F.R.C.S.

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Before commencing to examine the proper subject of this paper, I would desire to say a few words concerning the classification of hydrocele of the testis and cord, since the definition of the several subdivisions as at present given in text-books tends somewhat to mislead.

Classification should be based upon some definite principle, and that which appears to me both most natural and most convenient is the anatomical; I would therefore adopt a simple classification, based, like that of hernia by Mr. Birkett, upon the anatomical development of the part, and from which the disease is a divergence. With such a classification the name applied to any particular variety of hydrocele should explain the variety spoken of.

Following out this idea I would enumerate the four following varieties:

1st. Hydrocele of the tunica vaginalis, which may be either visceral or parietal.

2nd. Hydrocele of the whole vaginal process of peritoneum, which is necessarily congenital.

3rd. Hydrocele of the funicular portion of the vaginal process of peritoneum, which may be either congenital or acquired.

The acquired variety, varying in size, has unnecessarily received different names, according as to whether it be large or

small. When large it has been called diffused, when small encysted; these names, when applied to only one variety, varying in size, it becomes difficult to say when one is large enough to be called diffused, and when small enough to be encysted, as the latter can become the former by the pressure of the serous fluid opening up the areolar tissue, which feebly unites the two surfaces of the formerly open funicular process of peritoneum.

4th variety.—To this I would reserve the term encysted. It may be due to the dilatation of spaces in connective tissue, to the dilatation of the obliterated remains of the Müllerian duct, or of the organs of Giraldès, and to any other cysts occurring external to the tunica vaginalis testis.

Hydrocele of the tunica vaginalis signifies an undue collection of that serum which normally exists in the serous sac enveloping the testis, and which allows for any movement of the testicle, and keeps the two surfaces of the tunica vaginalis lubricated during the alterations in size, which necessarily take place during seminal secretion, as well as acting as a protection against external violence. The serous sac is formed by a prolongation of peritoneum, previous to the descent of the testis, which subsequently coming down posteriorly is suspended in the pouch, and becomes enveloped by it on nearly all sides. This tubular prolongation becoming finally closed from above downwards, a sac results at the lowest part.

As the lining of this sac envelops the testis on the one side, it may be described as consisting of visceral and parietal portions, the opposed surfaces of which are kept continually moist during health by a continuous serous secretion; the fluid thus constantly secreted is as constantly reabsorbed into the circulation, and it is only when this finely adjusted balance becomes unequal that hydrocele results. The parietal portion forms a blind pouch above the testicle, and for about the space of three quarters of an inch envelopes the spermatic cord, covering it rather more on the inner than the outer side. Posteriorly it becomes the visceral portion by being reflected along the back part of the epididymis, a small portion of which remains uncovered where the vessels and duct enter or emerge.

The visceral portion envelopes both the body of the testicle and the epididymis, and on the outer side is reflected into the

small digital fossa which exists between them. Below the testicle, and on each side of it the membrane becomes continuous with the parietal portion.

In connection with the visceral portion, a fact of great importance should be mentioned, for which we are indebted to M. Gendrin, who at page 64 of his work, 'Histoire Anatomique des Inflammations,' uses these words :

"The constant participation of the subserous cellular tissue, with the inflammation of serous membranes, can be explained by the fact that the inflammation extends itself to the parts which it covers, and reciprocally ; it also affords a reason for the limits that these inflammations meet with in their propagation. This proposition seems paradoxical, but it is nevertheless the consequence of facts.

"When the subserous cellular tissue extends into the contiguous organ, and penetrates into its thickness, one perceives that it becomes the means of communicating the inflammation ; whilst the propagation of the inflammation will not take place, however, when the contiguous organ shall be of different texture to that of the cellular tissue, or when this tissue shall be very different in the texture which it furnishes to the organ." Which, applied to the tunica vaginalis as to other serous membranes, means that where in connection with the epididymis the subserous cellular tissue penetrates and becomes continuous with that binding together the constituents of the epididymis, it readily becomes the means of communicating inflammation to the epididymis itself ; but where in connection with the tunica albuginea, which covers the body of the testicle, and not the epididymis, inflammation by contiguity does not take place. But, at the same time, to suppose that no areolar tissue exists between the body of the testicle and the tunica vaginalis is a mistake, as the base line of all serous tissues will show, that although small in comparison to that separating the epididymis, it is nevertheless sufficient to be easily affected by inflammation, whether it be from without or within. It is not the presence or absence of areolar tissue that limits the extension inwards of the inflammation to the body of the testicle, but the unyielding structure of the tunica albuginea, which resists inflammation from without. However, when the tunica albuginea is affected with inflammation, it is as liable to transmit it to the tunica

vaginalis, through the subserous cellular tissue, as the epididymis is; also the penetration of the areolar tissue among the interstices of the epididymis, making it the means of more ready communication of inflammation when the tunica vaginalis is primarily affected. Evidence in proof of this is frequently seen in disease.

The hydrocele in syphilitic orchitis is the result of the extension of the inflammation from the fibrous tissue, the tunica albuginea, to the adjacent tunica vaginalis; so, also, the hydrocele in gonorrhœal epididymitis is the result of the extension of the inflammation from the semi-muscular canal of the epididymis, both being examples of visceral hydrocele, or hydrocele produced by inflammation, extending from the body of the testicle in the one case and from the epididymis in the other to the tunica vaginalis.

On the other hand, the susceptibility of the epididymis to become indurated, as a secondary consequence to inflammation of the tunica vaginalis, led M. Panas, in the 'Archives Gén. de Méd.,' to believe that simple hydrocele was always caused by a partial epididymitis; and orchitis, as the secondary consequence of an inflamed tunica vaginalis, does not take place, for reasons previously mentioned. The sac of the hydrocele is usually thin, but in old and chronic cases it becomes thick, and, as the result of inflammation, lined by a red velvety false membrane, with occasional membranous bands and septa passing from one side to the other, thus accounting in some cases for the want of translucency.

Having described the serous sac itself, it remains to speak of its contents. Excessive accumulation of serum may be due to excessive secretion, passive exudation, or to defective absorption, originating from some cause which in its origin may be either inflammatory or non-inflammatory, and the hydrocelic fluid must consequently vary in its chemical composition. If inflammatory the serum will contain fibrine in solution; if merely dropsical the liquid is merely serous, and of a specific gravity of 1024 to 1028; the fluid varies in character from that of the serum of the blood to one containing more or less fibrine, and frequently some cholesterine, especially in old and chronic cases. An analysis quoted by Mr. Curling gives—

Water	.	.	.	.	91.25
Albumen	.	.	.	.	6.85
Uncoagulable matter	.	.	.	.	1.1
Salts	.	.	.	.	.8
					100.000

The colour varies from a straw- and lemon-yellow to amber and yellowish-red, which may be clear or more or less opalescent, the turbidity varying according to the amount of fatty molecules it may contain. The reddish colour which is sometimes imparted to the fluid is due to a slight admixture of blood, and the opalescent appearance to crystals of cholesterine. As much as six quarts have at one time been extracted from a hydrocele, viz. that of the historian Gibbon, by Mr. Cline, quoted in Sir A. Cooper's work 'On the Structure and Diseases of the Testis,' page 251. The average quantity, however, is about six ounces.

Passing now to the varieties of hydrocele of the tunica vaginalis, I would divide them into two, viz. visceral and parietal. This division I consider of great importance, as the prefix of either of these words at once signifies the origin of the hydrocele, and also gives a clue to the line of treatment to be adopted. A hydrocele which is visceral in its origin requires the treatment of the original cause, and not of the hydrocele itself, which is only secondary, just in the same way as a dropsy of the extremities is often secondary to disease elsewhere, say of the kidneys, heart, liver, &c., in which case the original cause, and not the consequence, has to be the subject of treatment.

Without entering into the question whether tapping is justifiable in gonorrhœal epididymitis associated with hydrocele, I would say broadly that when hydrocele is secondary to another disease the treatment of the hydrocele must and ought to be subsidiary to the treatment of the original complaint. I consider tapping in visceral hydrocele following epididymitis is as unjustifiable as it is in hydrocele following syphilitic orchitis.

Of visceral hydrocele the following are examples—that following epididymitis, that following syphilitic orchitis, and that following cancerous enlargements of the testicle or other forms of hydro-sarcocele; in these cases the visceral layer of the tunica

vaginalis becomes affected by contact with the inflammation of the epididymis or the body of the testicle, as the case may be, and as a result effusion takes place into the tunica vaginalis.

Of parietal hydrocele it is more difficult to get a simple example, as the visceral portion is probably also at the same time affected, or will soon become so by the continuity of inflammation; but in this form the body of the testicle will never present any sign of disease, nor will the epididymis, except secondarily as the result of means used to effect the cure of the disease by inflammatory action.

The following effects caused by a blow afford a good example of this variety, and show one of the chief uses of the serous sac of the tunica vaginalis in acting as a buffer to withstand external violence, which might be applied to the extremely sensitive organ the testicle.

It occurred in a young man hit by a cricket ball, who suffered no pain and had no enlargement of his testicle, but hydrocele resulted in consequence.

I believe a blow upon the scrotum will produce parietal hydrocele, whilst a blow in excess of the expenditure of force required to cause hydrocele will produce orchitis.

Here as the result of the blow an excessive secretion of serum takes place, which distends the sac of the tunica vaginalis, and one of the forms of traumatic hydrocele is produced.

Traumatic hydrocele may also occur from a secondary inflammation of the tunica vaginalis following orchitis. Therefore traumatic hydrocele occurs on the side upon which the injury is received, and differs from idiopathic hydrocele, which is most frequently met with on the left side.

Parietal hydrocele may be also caused by the irritation of loose cartilaginous bodies consequent upon the rupture of the peduncle of the hydatid of Morgagni (see p. 81 of vol. v of 'St. Thomas's Hospital Reports').

Perhaps the best example of parietal hydrocele is that occurring in old people which is chiefly caused by passive exudation, resulting from the pendulous condition of the scrotum and due to the relaxation of the capillaries of the tunica vaginalis consequent upon the general arterial relaxation of old people, whereas hydrocele of the tunica vaginalis in young adults, except arising from injury, is essentially of the visceral form.

Hydrocele of the tunica vaginalis may occur on either or both sides, the relative frequency of the two sides being according to Curling in favour of the right side; but I am inclined to agree with Bryant, Velpeau, Gerdy, and Dujat, in believing the left side to be that most frequently affected, as during the time of my registration of cases at the hospital I carefully collected the notes of some years which show a little in excess of one half to be on the left side, one quarter on the right, and a little below one quarter double. The numbers are thirty-one on the left side, fourteen on the right, and eleven double.

In these statistics I have been careful to exclude all cases of traumatic hydrocele, and believe that if all authors classified only idiopathic hydrocele their statistics would also show a preponderance in favour of the left side.

The greater frequency of the left side is, I believe, due to the greater dependency of the left testis and the mode of exit of the blood into the renal vein, producing venous congestion and subsequently excessive transudation of serum. The question why the left testicle hangs lower than the other has never received a satisfactory solution. That it is the first to enter the scrotum is possibly the reason it is ultimately situated the lower, but why it should enter first is not accounted for by the fact that it has the longest way to travel, but possibly to the fact that in the process of the development of the great blood-vessels and the obliteration of the arches on the right side, the left becomes more fully supplied with blood and therefore precedes the right in its developmental activity.

Anyhow the chief cause of hydrocele is undoubtedly venous congestion determined by hepatic or renal enlargements or any obstruction to the outflow of blood, not omitting inguinal hernia, which Mr. Curling, observes to be "a disease obviously very favorable to the effusion of serum in the tunica vaginalis, owing to the pressure of the rupture on the veins of the spermatic cord, and which is often increased by the use of trusses and bandages."

Its greater frequency amongst inhabitants of warm climates, especially of the West Indies, is probably due to venous congestion arising from hepatic enlargements, which we know to be so common amongst those inhabitants.

Hydrocele of the tunica vaginalis is most frequently met

with in middle-aged persons between the ages of forty and fifty ; occasionally, however, in infants, but probably in these cases they are truly hydroceles of the vaginal process of peritoneum, there still remaining some patency of the opening into the peritoneal cavity, as the opening in the funicular portion may be small enough to allow of fluid percolating drop by drop into the tunica vaginalis, but yet not large enough to allow of its being returnable into the peritoneal cavity, the diagnostic symptom of hydrocele of the vaginal process of peritoneum. Amongst old people the disease is as prevalent as amongst the middle aged, for the proportion of old people living affected with hydrocele is as great as the proportion of middle-aged people affected with the same.

As to the effect of hydrocele upon the functions of the testicle, it seems a large hydrocele is likely to cause the suppression of the spermatic function on the side upon which it is situated, from the long-continued pressure of the fluid and by the stretching of the epididymis from the body of the testicle ; therefore double hydrocele, under certain conditions, should be enumerated as one of the causes of sterility in the male. These remarks apply only if the disease has been allowed to continue for any length of time, and therefore afford a great reason for early treatment. A paper on this subject was communicated by M. Lannelongue to the Société de Chirurgie, July 16th, 1873, and reported in 'L'Union Médicale.' The author there states that in large hydroceles no spermatozoa were found either in the epididymis or vesiculæ seminalis ; whereas small hydroceles do not lead to complete suppression of spermatozooids, but the animalcules are modified and altered in the seminal passage. He therefore considers that early treatment is called for in cases of hydrocele, in order to prevent the weakening and abolition of the spermatic functions.

Hydrocele in the female, or a collection of serum in the lower part of the canal of Nuck, the analogue of the vaginal process of peritoneum in the male is occasionally met with ; of such I have been fortunate enough to see an example, and as the rarity of these cases is great the notes are here subjoined.

"E. D—, æt. 45, married, with two children, was admitted into the hospital on May 29th, under Mr. Wagstaffe. She first noticed a lump on the left side in the position of the inguinal

canal about nine years ago, and which in size was about that of a gooseberry. She was in the habit of pushing it up, but it seldom remained so, and she finally wore a truss, believing it to be a rupture ; it always remained up when lying down.

"The tumour had grown much larger the last two years, principally, she thought, through having to do a good deal of standing about, and on admission was the size of three walnuts, very tense, not painful, evidently with fluid contents, not returnable into the abdomen, with no sickness or abdominal tenderness.

"The left hip-joint had been dislocated since childhood and ankylosed.

"30th.—Chloroform given and cyst punctured with fine trocar and about three ounces of reddish-yellow fluid withdrawn. Four days afterwards cyst was found to be refilling, and on June 10th the cyst was retapped, and after about two ounces had been withdrawn, one drachm of Tinct. Iodini was injected.

"The cyst subsequently refilled, but finally diminished, and she was presented cured on June 24th."

The absence of pain and impulse on coughing, with no disturbance of digestion, and the elongated form of the swelling, extending along the inguinal canal, and transparency, are the means of diagnosis from hernia.

The diagnosis of a hydrocele may be considered under the following headings:—Scrotal swelling, fluctuation, transparency, testicular sensation.

The presence of a scrotal swelling which on examination is found to be internal to the testicular coverings and external to the testicle (the testicle being in no way implicated, and in addition placed posteriorly and to the upper part of the swelling) points to a collection of fluid in connection with the tunica vaginalis which can be only one of two things, hydrocele or hæmatocoele, and which if translucent can be definitely affirmed to be the former, but if opaque the diagnosis is one of extreme difficulty and can only be decided by the trocar.

For hydrocele and hæmatocoele may both have in common the following symptoms—want of translucency, fluctuation, testicular sensation, sudden appearance and history of injury ; so in forming an opinion it behoves one to speak with great caution,

for the shades of gradation from haematocele to a hydrocele containing a certain amount of blood are so gradual that any one speaking positively will frequently find himself in error. Especially is this likely to occur in cases of haematocele following hydrocele, but the sudden appearance of the swelling, history of injury with ecchymosis of the scrotum, and solidity, are points in favour of its being haematocele. An amendment should also be made as to the position of the testicle being always posterior; it is rarely found anterior, and when this occurs it must be accounted for by some abnormality in the descent of the testis at birth; but not unfrequently the testis is irregularly placed, owing to adhesions taking place between the two layers of the tunica vaginalis.

The size of this scrotal swelling will vary from that of a hen's egg to one of such magnitude that the penis is completely buried and merely represented by a button-like aperture, and is annoying to the patient in not only preventing the proper functions of the organ, but also, on account of size, clumsiness, and an inability of secreting it, and although no actual pain is occasioned, a feeling of weight and a dragging sensation along the cord is experienced. Its shape is oval in recent cases, pear-shaped in old ones, owing to the patency or the pressure of the hydrocelic fluid opening up the funicular portion of the tunica vaginalis. It not infrequently presents an hour-glass shape, owing to the natural contraction of the tunica vaginalis above the testicle or to the presence of a hydrocele of the funicular portion of the vaginal process opening into a hydrocele of the tunica vaginalis. The shape is, moreover, often irregular owing to the adhesion of the visceral and parietal portions of the tunica vaginalis together, "the outline of a hydrocele depending upon the anatomical conditions of the part in which it is situated and the pathological changes which may have resulted from the affection."

Fluctuation detected by palpation is a very important symptom in the diagnosis of hydrocele.

Translucency is, however, the chief aid to a correct diagnosis, although it cannot be solely relied upon, for transparency is at once indicative of some form of hydrocele, and should any malposition of the testicle be present this is at the same time seen and remembered in the course of treatment. Complete

translucency is spoken of by some French writers, but such a phenomenon is impossible unless in a case of undescended testicle.

Transparency is conclusive evidence in favour of one of the forms of hydrocele; but, on the other hand, its absence does not militate against its being a hydrocele, as an opaque fluid or a thick-walled sac will falsify the result. The best means for detecting transparency is by using a stethoscope applied firmly to the scrotum on the one side, the integument of which is tightly stretched by the left hand placed posteriorly, whilst a lighted candle is closely applied on the opposite side; the hand is frequently used as a tube, but imperfectly so, as rays of light cannot fail to pass between the fingers and render the transmission of light imperfect. Testicular sensation is that peculiar sickly feeling which results from squeezing a healthy testicle, and is always to be found in parietal hydrocele, but is frequently absent in visceral hydrocele, that is, when the hydrocele is secondary to some form of orchitis. On manipulating a hydrocele this should, if possible, be determined, as marking the position of the testicle will prevent any injury thereto in a subsequent tapping.

The cure of a hydrocele is effected in one of two ways, either by the simple withdrawal of the excess of serum, with or without the injection of mild styptics, whereby a fresh and healthy action is imparted to the tunica vaginalis and secretion and absorption are carried on reciprocally; or by the obliteration of the sac, either by the injection of strong styptics, whereby adhesive inflammation is set up and the sides of the tunica vaginalis become more or less approximated, and adhesions take place between the visceral and parietal layers, or by allowing the sac to suppurate and granulate up from the bottom.

Before commencing a description of the several modes adopted for attaining either of the above ends, it should be borne in mind that, according as constitutions vary in their degrees of stability, so in the same ratio will the effects of inflammation vary in those same constitutions; I propose, therefore, to consider the modes of treatment *seriatim*, taking the milder measures first, and I would lay down as a general rule of treatment that it is not advisable to resort to severe

measures to attain a result when milder ones will answer the same purpose.

In children the application of the ordinary evaporating lotions frequently succeeds in effecting a cure, probably due materially to the good feeding or tonic treatment with rest resorted to at the same time; spontaneous disappearance is not uncommon in children, and has been spoken of in connection with adults. I have never been so fortunate as to have seen an example, but Bryant, in his 'Practice of Surgery,' cites one case which he believes to have been one; but some of the cases quoted of spontaneous disappearance are ones actually cured by inflammation which has been set up by some accidental violence, or by inflammation extending from the urethra to the testicle, and thence to the tunica vaginalis. One case of spontaneous disappearance cured by inflammation resulting from external violence is quoted by Sir A. Cooper at page 257 in his work on 'The Structure and Diseases of the Testis.' The external application of iodine, blistering fluid, and mercurial inunction, have occasionally proved successful remedies in recent cases, but so rarely, and with, in addition, so much discomfort and pain, that it is but seldom resorted to.

Acupuncture is eminently successful, especially in young children; the hydrocele is punctured in two or three places with a broad needle, the flat of the needle being turned at right angles to the puncture before being withdrawn, allowing the hydrocelic fluid to percolate into the cellular tissue external to the tunica vaginalis, substituting "an anasarca of the scrotum for a dropsy of the tunica vaginalis," the fluid being subsequently removed by absorption.

The cure results from the same cause as that by tapping, the withdrawal of the fluid allowing the tunica vaginalis to recover its lost balance between secretion and absorption. To say that the cure of the disease in these cases is effected by the support and compression afforded to the vaginal sac by the infiltration of the surrounding cellular tissue is, I believe, a mistake, as I have never yet known a hydrocele cured by compression.

Acupuncture is a mode of treatment which is simulated in nature by the accidental rupture of the tunica vaginalis and escape of the hydrocelic fluid into the surrounding cellular tissue,

for when a hydrocele is of large size jumping from a height or a blow or bruise will readily burst it. This is followed by œdema of the penis and scrotum, and affords a striking analogy to what takes place when in the injection of iodine some of the injection is inadvertently thrown into the surrounding cellular tissue external to the tunica vaginalis. Such an accident as the latter may be due to the gradual instead of the sudden thrust of the trocar into the distended sac, the parietal portion being thereby separated from the scrotal tissues, a result which may also take place from an imperfectly fitting trocar and canula.

The result which follows such an injection of iodine into the cellular tissue is, I believe, exemplified in the case quoted by M. Gendrin at page 143 of his work on 'Inflammations,' where gangrene resulted in consequence. Simple tapping will sometimes effect a cure; at other times, however, the fluid will re-collect, but after repeated tappings at increasing intervals an ultimate cure is established. It is said that this mode of treatment is most successful in former inhabitants of the West Indies, but it is not at all uncommon to find examples amongst others; in old people it is as well to temporise with tapping, as they are seldom able to stand more severe measures.

The position of the testicle having been first made out by the transmission of light or by manipulation, a well-fitting trocar and canula slightly oiled, with the finger placed about one inch from the point to prevent its being pushed onward too far and wounding the testicle, is quickly thrust, avoiding any superficial vein which may be perceptible, anteriorly through the scrotal coverings, which are rendered tense by being grasped firmly by the left hand; as the trocar is extracted the canula is pushed further home and the fluid withdrawn. Should any thickening of the testicle remain after the withdrawal of the fluid, the application of strapping should be employed.

After the withdrawal of the canula no application to the puncture is required, as the collapsed tunica vaginalis will fall as a valve over the opening, and if the scrotum has not been over-distended for some time, so that the muscular tissues be paralysed, the contraction of the dartos will also assist in its closure.

In tapping, then, the following things have to be avoided—  
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wounding the testicle, puncturing any superficial vein, injuring the spermatic vessels situated posteriorly, and separating the parietal layer of the tunica vaginalis from the scrotal tissues by slow puncture.

Tapping may be performed with supplemental means, such as irritating the internal surface of the tunica vaginalis by scratching it with the end of the canula, or by passing up the canula a probe coated with nitrate of silver and applying this to the surface of the tunica vaginalis, the latter being the better operation of the two and frequently productive of the best results.

A somewhat similar plan has been tried by Italian surgeons in the application after tapping of the negative pole of a battery to the inner surface of the tunica vaginalis, the positive pole being applied to the external surface of the scrotum.

Injects may also be used in connection with tapping, of what description is a matter of taste, any irritating fluid which is of sufficient strength to set up some inflammation of the tunica vaginalis answering the purpose. Cold water, milk, port wine, spirits of wine, a solution of alum or sulphate of zinc (3 grs. to 3j), or tincture of iodine, have all been used with different results, port wine in the hands of some surgeons having succeeded where iodine has failed. The last-named remedy is the most popular since the excellent results obtained by Sir R. Martin in Calcutta. However, to place this treatment under the head of radical, and to leave out milder remedies, is to say that milder means are never radically curable, and that this mode of treatment never fails, both of which statements are incorrect.

I find on examining the records of old cases of hydrocele treated by injection of iodine that out of fifty-four cases nineteen had been previous iodine failures. Of these fifty-four treated by tapping and injection I have made inquiry, but from twenty-five only have I been able to get replies, and I find that of the twenty-five only seven have had no recurrence, whereas eighteen have recurred. It is further noticeable that of these eighteen two had failed once before.

Of iodine injected some prefer a solution, others 3ij of the pure tincture; whichever is used does not apparently much matter, the chief point being the manipulation of the sac so

that the injection may be brought thoroughly in contact with the whole of the internal lining of the tunica vaginalis.

The amount of pain consequent upon the injection cannot be taken as a criterion as to the ultimate result, for facts show that not infrequently the best results are obtained in cases of the least suffering.

The operation should only be performed at the patient's own house or where no exertion has to be taken subsequently, as occasionally severe inflammation has been set up in consequence.

The subsequent treatment is also one of great importance, the patient being confined to his bed for three or four days, and the testicle supported and ice applied if pain or inflammation be excessive.

Another point of importance should be remembered, and that is that a platinum canula be used, as the ordinary silver canula is destroyed by the action of the iodine.

If port wine be chosen, about six to eight ounces pure are injected and manipulation used as in other cases, and after having been allowed to remain in from ten to fifteen minutes withdrawn. In injecting such a large quantity there may enter not infrequently a certain amount of air which gives the characteristic crackling sensation of emphysema, but this result is, however, of no great importance, and the air is soon absorbed.

The injection of a mixture of equal parts of carbolic acid and glycerine has been tried by Dr. Lewis, of Philadelphia, who considers it more certain in its action and less painful than iodine.

Under the head of secondary consequences, besides emphysema previously mentioned, there are refilling of the sac, orchitis, and gangrene.

Besides the refilling of the sac and failure of the treatment, there is another form of refilling not unfrequently met with, which is subsequently followed by reabsorption and ultimate cure, consequent upon the pouring out of inflammatory products; therefore in cases where a refilling of the sac takes place it behoves one to speak with caution as to the ultimate result.

Orchitis following injection is due to a primary epididymitis extending to the body of the testicle, in the same manner and

for the same reasons as was previously mentioned in the extension of disease from the epididymis to the visceral layer of the tunica vaginalis.

Gangrene seldom occurs if the operation be performed in a proper manner and upon fit subjects, as the result of the injection of iodine into the cellular tissue between the tunica vaginalis and scrotal tissues I have previously mentioned, and in aged persons it is advisable in all cases to temporise with tapping alone.

Tetanus has also occurred, of which Mr. Curling gives several examples; it is, however, so rare an occurrence that it hardly enters into the category of probabilities.

As to the treatment by setons, I would refer to the remarks made by Mr. Green on this subject at p. 73 in the old series of 'St. Thomas's Hospital Reports,' the success of the cases there cited speaks in favour of this mode of treatment when held to be necessary.

The seton has been used in two ways, either by allowing it to come away by ulceration and consequently laying open the tunica vaginalis, or only leaving it in a sufficient length of time for inflammation to be produced; the latter is the usual mode, viz. after the hydrocele has been tapped at the lower part by a trocar and canula and the fluid withdrawn, a long needle coated with wax, to prevent injuring the testicle and armed with the seton, is passed up the canula to the upper part of the sac; the needle is then made to perforate the scrotal tissues, the two ends of the seton being subsequently knotted together on the withdrawal of the canula. This seton is left in from thirteen to thirty-five hours, or rather should not be withdrawn "until some indication of febrile action in the system has been experienced."

The severe symptoms during treatment by the seton were apparently due to the retention of inflammatory materials by the seton itself, as pus in all cases followed its withdrawal, an objection which would be materially obviated by the substitution of a drainage tube for the threads of the seton, as thereby the interior of the tunica vaginalis could be thoroughly washed out by syringing, and retention of inflammatory products rendered inevitable.

Excluding accidental rupture, which is virtually an operative

procedure, a natural cure has rarely taken place, but of such, from excessive distension, Sir Astley Cooper gives an example at p. 256 in these words :—"A slough of the scrotum and tunica vaginalis is produced, and as it separates the water escapes, a suppurative inflammation succeeds, granulations arise, and in this way the hydrocele becomes spontaneously cured ;" this is, however, too severe a proceeding to justify any surgeon in allowing things to proceed so far ; it is, however, a process which has been simulated in the treatment by caustics, the continued application of which to the scrotum has produced a slough through to the tunica vaginalis, on the separation of which the serum escapes and the hydrocele is finally cured by adhesion or granulation.

The frequent application of the caustic, the painfulness of the operation, and the severe effects set up in consequence, would hinder any surgeon of the present day performing, or any patient submitting to, so severe a procedure ; more especially will the same remark apply to the two following methods, which are more severe, viz. incision and excision.

Incision, which is solely applicable to those cases where the presence of loose cartilaginous bodies are the exciting cause of the hydrocele, or should any doubt exist as to its being a hernia, or as an aid to diagnosis previous to performing castration, should then be performed with a limited incision. The old method, the chief advocate of which was Mr. Bell, is performed in the following manner :—The external integuments are divided by one continued incision from the upper to the lower end of the tumour ; an opening at the upper end of the incision is made into the vaginal coat large enough to admit the finger, which is passed down as a director for the bistoury and the tunica vaginalis laid open to a corresponding extent with the primary incision. The tumour being opened from above, the hydrocele does not collapse and consequently lessens the risk of injuring the testicle, the cavity of the tunica vaginalis being subsequently dressed with (carbolized) oiled lint, in a similar way as in the treatment of bursæ, thus allowing it to heal up by granulation, a mode of treatment which not unfrequently terminated fatally. Professor Volkman has since modified the seriousness of this operation by performing it antiseptically, and has had some good results, so also have

those surgeons who have performed the operation in the same way in England; therefore, when incision is held to be necessary, the antiseptic method should be adopted.

Excision consists in the removal of more or less of the parietal layer or both layers of the tunica vaginalis after it has been gradually cut down upon; but this operation requires to be mentioned only to be deprecated, and is only resorted to when less severe measures fail, which is but seldom.

## INTRACRANIAL ANEURISMS.

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MORGAGNI, in alluding to the death by apoplexy in 1714 of Bernadin Ramazzini, suggests that the symptoms might have been caused by the rupture of an aneurism of the arteries of the brain; founding this opinion upon the circumstance that Ramazzini had for some years had a small aneurism in each hand between the finger and thumb. It does not, however, appear that Morgagni had ever seen any case of the kind, though he describes aneurismal dilatations and other morbid changes in the coats of branches both of the internal carotid and vertebral arteries. In 1765 a case of aneurism of the internal carotid artery in a female of advanced age, who had suffered from severe rheumatic attacks, was published at Milan by Biumi and was reprinted by Sandifort at Leyden in 1778. In 1800 Sir Gilbert Blane described the case of a lady, æt. 64, who had been suddenly seized with dimness of sight and acute pain in the forehead which continued for some months; two years after she had a similar attack followed by mania, which was relieved, but recurred, and she continued to suffer until her death, which took place in her 69th year. The post-mortem examination was performed by Mr. Hunter, assisted by Mr. Home, and in the presence of Dr. Jenner and Sir G. Blane. In addition to the morbid conditions in the

brain, the internal carotid arteries, where lying in the groove on each side of the sella Turcica, were very greatly dilated, that on the left side being the larger of the two. Reference is made to this case by Dr. Baillie in his 'Morbid Anatomy.'

In the 'Anatomy of the Human Body,' by John Bell, the case is related of a young woman, who after a slight accident was taken with apoplectic symptoms, and it is stated that the internal carotid artery was found torn across, but no mention is made of the vessel being dilated or otherwise diseased at the seat of the rupture.

In 1814 a case of aneurism of the basilar artery in a young female, which had ruptured and produced fatal coma, was published by Dr. Blackall in his work on *Dropsy*; and in the following year the same case was more fully recorded by Mr. Hodgson, with the statement that the preparation had been sent to him by Mr. Barnes, of Exeter. Mr. Hodgson also describes a second case in which an aneurism of the anterior cerebral artery had undergone a natural cure, the sac being obliterated: the patient was an incurable lunatic who had been seventeen years in Bethlem Hospital. This specimen was then in the possession of Mr. Langstaff, and is now in the museum of the Royal College of Surgeons. Mr. Hodgson refers also in a note to a third case as having fallen under his notice after the former part of his work was printed. The sac was situated on one of the arteries of the cerebrum, and was as large as a cherry; it was empty and had ruptured.

In 1825 a case of aneurism of the anterior cerebral artery was published by Mr. Spurgin, of Saffron Walden. It occurred in a man aged fifty-seven, who was suddenly seized with apoplectic symptoms. After a partial recovery and relapse he had a third fit in which he died. The aneurism was about the size of a hazel-nut and had ruptured.

In the following year two cases were published by M. Serres, though it appears that the cases had previously appeared in 1819 in a journal to which I have not been able to obtain access. Serres' cases are frequently referred to as those of Bouillaud, that writer having referred to them in a special memoir on apoplexy from disease of the cerebral vessels. In the first of the cases a man, aged 59, died suddenly during convalescence from an attack of pneumonia, and an aneurism of

the basilar artery, the size of a small fowl's egg, was found ruptured. In the second case, a female, aged 53, had epileptic seizures, and was paralyzed on the left side more especially, and an aneurism the size of a musket ball of a bilobulate form was found to occupy the anterior communicating artery, and one side of the aneurism had burst.

In 1827 Mr. Chevalier published a case in which a tumour was found between the sella Turcica and brain which was regarded as aneurismal. The subject of the disease was a female, aged 37, who had been subject to headaches from infancy. About six months before death the pains became much more severe and shot through from the back to the right eye; the sight became affected and she was occasionally dizzy. She had a fit in which she fell unconscious in the street three weeks before death, and she died comatose. There was a limited extravasation of blood in the left parietal region and a tumour the size of a large walnut was firmly attached to the sella turcica. It contained layers of discolored coagula with fluid blood in the cavity. It was supposed to have arisen from the left anastomosing artery of the circle of Willis. Soon after this case was published a case occurred at St. Bartholomew's in a patient who had had a slight paralytic stroke two years before and died of fever, an aneurism the size of a nutmeg was found in the middle cerebral artery, immediately before its origin from the internal carotid.

In 1829 Mr. Gore recorded a case in which an aneurism of the left vertebral had ruptured and occasioned an apoplectic attack fatal in about twenty-four hours. The patient was a man of twenty-four, and the sac was only of small size. In the following year a case occurred in the practice of Dr. John Bright at the Westminster Hospital; the subject of the disease was a man, æt. 35, who had been paralyzed for about two years. The aneurism was situated on the basilar artery, and was about the size of a chestnut, the sac was not ruptured and it had compressed the pons.

In 1831 a case appears to have occurred at St. Thomas's in which an apoplectic attack was traced to the rupture of an aneurism of the internal carotid artery immediately where it gives off its branches; and in the same year a case is recorded as having occurred at the Hôtel Dieu to Dr. Gueneau

de Mussy in which an aneurism in a very similar position produced fatal apoplectic symptoms in a man aged 60.

Dr. Richard Bright, in the second volume of his 'Reports,' published in 1831, has recorded two cases of cerebral aneurism. The first of these occurred in the practice of Mr. Streeter. A man, *æt.* 19, was suddenly taken with pain in the head and coma; he partially recovered, but died in a similar attack about a week after. Extravasation of blood was found on the left hemisphere, derived from rupture of an aneurism of the left middle cerebral artery. In the second case, a man, *æt.* 45, was taken with apoplexy in Guy's Hospital, and died in thirteen hours, and there was found a large extravasation of blood and an aneurism of the left middle cerebral artery nearly an inch in one diameter and rather more in the other. The vessel leading to the aneurism was obstructed by clot.

In 1832 and 1833 a notice of a case which occurred in the practice of Mr. Jennings, of Leamington, was published, and in 1836 the same case was detailed more fully. The case was that of a man, *æt.* 54, who had served for some years in the army; he was suddenly seized with sense of suffocation, and pain at the back of the neck and drawing back of the head, followed by partial coma, and died shortly after. A large effusion of blood was found over the *tuber annulare* and *medulla oblongata*, and extending down the whole length of the spinal canal. The blood was found to be derived from the rupture of an aneurism about the size of a pea in the *basilar artery*.

In the same year M. Flandin exhibited at the Soc. Anatomique of Paris a specimen of a small aneurism of the right middle cerebral artery which had ruptured, producing left hemiplegia in a man, *æt.* 60. The case occurred at the Hôtel Dieu.

About this time Breschet referred to a case which had occurred to M. Amussat, and this case has sometimes been referred to as Breschet's, and at other times as Amussat's case. It was a fusiform aneurism of the internal carotid in the cavernous sinus, and occurred in a man, *æt.* 50.

In the following year an aneurism of the artery of the corpus callosum (anterior cerebral), which was found in a married female, *æt.* 28, was made the subject of a graduation thesis published at Heidelberg in 1834 by Dr. J. H. Nebel. The

tumour was about the size of a walnut ; it had compressed the left optic nerve, and caused loss of sight of the right eye.

In this thesis the cases of aneurism of the cerebral arteries were first collected together. In addition to the foregoing case he here mentioned twelve others, including those of Gilbert Blane, the doubtful case of John Bell, the cases of Hodgson and Serres, one published in the 'Lancet,' and Chevalier's, Jennings', and Breschet and Amussat's cases, together with one of aneurism of the middle meningeal artery published by Krimer. In the following year a memoir on the subject was published by Professor Albers, of Bonn, in which fourteen cases are collected, the two cases not included in Nebel's collection being that of Spurgin and that which had been published in the previous year by Bergmer, one of Serres' cases being here referred to as that of Magendie, and Krimer's case being still included in the enumeration. The case mentioned by Albers as published by M. Bergmer appeared in a thesis. It is that of a female, æt. 60, who died rapidly with cerebral symptoms from the rupture of an aneurism the size of a hazel-nut, which involved the internal carotid and its branches.

In the year 1835 Mr. King published three cases which had occurred at Guy's Hospital. The first of these occurred in a man, æt. 45, who died of apoplexy after having had several premonitory attacks, and there was found an obstruction by clot of the left middle cerebral artery, and a tumour upon the trunk of the right middle cerebral artery half an inch long and rather less wide.

In the second case a man, æt. 30, died comatose very rapidly, and there was found after death blood in the lateral and fourth ventricles which had escaped from a bilobate aneurism of the right anterior cerebral at its point of bifurcation.

In the third, a female, æt. 56, after an apoplectic attack was left paralyzed on the right side with loss of power of speech, and six months after had another attack in which she died in three weeks. A cyst was found in the left hemisphere, and some recent extravasation in both cerebral hemispheres. There was a minute aneurism in the fissure of Sylvius, which, however, had not ruptured.

In 1836 two cases are recorded, one by Lebert, in the

‘*Bulletin de la Soc. Anat.*,’ in which an aneurism the size and form of a hen’s egg arose from the basilar artery, and had compressed the adjacent parts of the brain and the nerves. It occurred in a man, aged sixty-eight, who was first paralyzed in the lower and afterwards in the upper extremities also, and he had both aphonia and dysphagia and attacks of dyspnoea, in one of which he died. The other case is contained in a graduation thesis published by Stumpff, at Berlin in 1836. This thesis comprehends the fifteen cases, including those of Mr. King previously recorded. Stumpff’s own case is that of a man, æt. 22, who had suffered from symptoms of acute cerebral disease, and afterwards from pain in the right eyeball, and the globe was turned outwards, the pupil dilated and insensible, and the eyelid fell. He was taken with convulsions and coma, and died. There was much blood extravasated in the Sylvian fissure and base of the brain, and an aneurism was found arising from the angle between the Sylvian (middle cerebral) artery and the posterior communicating artery the size of a hazel-nut which had burst.

In 1838 and 1839 a case is recorded which had occurred to Dr. Burne at the Westminster Hospital, in which a man of thirty-five died with apoplectic symptoms produced by the rupture of an aneurism in the right middle cerebral artery. It was about the size of a swan shot.

The following year Mr. Porter, of Dublin, mentioned having seen an aneurism the size of a bean on the basilar artery in a patient who died comatose after having long suffered from urinary disease and impaired mental power.

In 1841 Engel treated a patient, æt. 17, who had an aneurism of the basilar artery as large as a plum.

In 1842 two cases are recorded. In one by Dr. Kingston, a boy fourteen or fifteen years of age, died after having had convulsions for a week. An aneurism the size of a walnut was found on the basilar artery. In another, by Dr. A. T. Thompson, a man, æt. 49, died with apoplectic symptoms, and there was much blood extravasated which had escaped from an aneurism a little larger than a hazel-nut. It arose from the left middle cerebral artery, where it divides into its branches. The third was described by M. Delpech in the ‘*Bull. de la Soc. Anat.*’

Two years later, or in 1844, three cases were related by Mr. R. W. Smith. In the first case the patient was fifty-four, and subject to violent attacks of mania and epilepsy, and he died comatose two days after a sudden attack, and an aneurism the size of a small apple was found occupying the floor of the third ventricle and compressed the optic and olfactory nerves of left side, and the tuber annulare was partially destroyed. It was supposed to have originated from a branch of the basilar. In the second case an aneurism 4 Paris lines long, 3 broad, and 3 deep, had ruptured, in a man, æt. 41, and in the third case an aneurism an inch long and half an inch across arose from the basilar artery in a man, æt. 40, subject to giddiness and weakness of heart and general paralysis.

In 1846 four cases were related, one by Ruschenberger of the U. S. Navy, in which a man died with paralysis of the left side, and an aneurism projecting from the right side of the basilar artery was found about the size of a pigeon's egg, which had pressed on the pons Varolii and ruptured into it.

The second case was a specimen exhibited by Dr. Eager at the Manchester Pathological Society.

The third was a specimen also exhibited at the Manchester Pathological Society by Dr. Francis. A female, æt. 62, died suddenly with coma and convulsions, and an aneurism was found which involved part of the left internal carotid and the anterior communicating artery. It was about the size of a bean and had ruptured.

The fourth case, by Mr. France, was that of a female, æt. 20, admitted into Guy's Hospital with symptoms of pressure on the third nerve, and who soon died comatose. An extravasation of blood of the right posterior communicating artery, supposed to be due to an aneurism, was found at the base of the brain.

In 1847 four cases are recorded. Of these two are related by Dr. Edwards Crisp. In one of these a man, æt. 35, after suffering for four months with pain in the head had a sudden attack and was paralyzed on the left side, and died in nineteen days; a small coagulum was found in the left lateral ventricle, the left corpus striatum was softened, and an aneurism the size of a hazel-nut was found to arise from the carotid artery at its point of division. In the other case a man, æt. 40, died suddenly, and an aneurism of the size of a walnut was found

to arise from the basilar artery, but no rupture had occurred. The heart was fatty, its walls thin, and the cavities dilated.

The remaining six of the eight cases referred to by Dr. Crisp are those published by Sir Gilbert Blane, Dr. Kingston, Dr. Thompson, Dr. John Bright, and Dr. Richard Bright.

Dr. Crisp also reported a case, that of a boy, æt. 14, a patient of Mr. Beane, of Peckham, in 1839, who died four days after an apoplectic attack, having previously suffered from giddiness. There was extreme extravasation on the upper surface of the brain and in the ventricles: two small aneurisms, one as large as a pea, the other the size of a bean, existed on one of the branches of the anterior cerebral artery, and the latter had burst.

In 1848 Dr. Hare and Mr. Moore reported cases. In the first of the cases, a girl of 18, an aneurism six tenths of an inch long and four tenths of an inch wide arose from the left posterior communicating artery and had ruptured. Two years before she had intense headache and delirium. She was taken suddenly with dimness of sight and paralysis of the muscles supplied by the third nerve of the left side, and died in three weeks.

In Mr. Moore's case the aneurism was noticed at the post-mortem examination, but there is no account of the symptoms. It arose from the external side of the internal carotid artery, compressed the optic commissure, and had led to absorption of the sphenoid bone. The sac had ruptured, and there was great extravasation. The patient was a female, æt. 52, who had an apoplectic attack, and died in a few hours at the Middlesex Hospital.

In 1849 six cases were related. In one of them (Dr. H. Douglas), a female, æt. 39, died apoplectic, and an aneurism of the anterior cerebral artery just before it divides, as large as a bean, was found to have ruptured and caused extravasation.

In another case, observed by Dr. Niell, a man who had an epileptic attack was found to have an aneurism as large as a filbert on the right middle cerebral artery.

Dr. Bowen described a third case in which an aneurism the size of a pea arose from the basilar artery, and had ruptured. The subject was a female, æt. 83, who was admitted into the New York Hospital and died comatose.

In a fourth instance, Dr. Gordon found an aneurism of small

size which had ruptured and produced apoplectic symptoms in a man, *æt.* 35.

In a fifth instance, M. Barth described the case of a female at the Saltpêtrière, *æt.* 70, who died with symptoms of subacute cerebral disease, having previously had ptosis and strabismus. An aneurism of the posterior communicating artery was found.

In a sixth, M. Boudet related a case under care of M. Petit at the Hôtel Dieu, a man, *æt.* 57, who had had cerebral symptoms for two and a half years, and died comatose, and an aneurism which had ruptured was found at the side of the internal carotid artery.

In 1851 Dr. Fletcher, of Birmingham, published the case of a man, *æt.* 35, who died suddenly from rupture of an aneurism of the middle cerebral artery.

In 1852 Dr. H. Roe exhibited a case of aneurism of the anterior communicating artery the size of a hen's egg, projecting into the anterior lobe of the right hemisphere and comprising the right crus and optic tract, in a female, *æt.* 21, who had suffered from violent headache for fifteen months before death. The aneurism was not ruptured.

In the same year Dr. Brinton, in reporting on this case in the 'Pathological Transactions,' appended a table in which he gave statistical details of fifty-two cases; of these, however, one, that of Krimmer, aneurism of the middle meningeal artery, as pointed out by Sir W. Gull, does not belong to the same category, and two others appear twice in the table as quoted from different sources; the enumeration is therefore reduced to forty-nine. Of these five have no references, and were probably communicated to Dr. Brinton by the gentlemen in whose practice they occurred.

In 1859 Sir William Gull contributed to the 'Guy's Hospital Reports' a paper in which he collected twenty-nine cases, of which eighteen had been included in Dr. Brinton's table; two cases were quoted from the 7th volume of the 'Pathological Transactions'; two were collected from other sources, and seven were new and original cases. In this paper Sir W. Gull entered more fully into the symptoms of such affections than Dr. Brinton had done, and endeavoured to establish means by which they might be diagnosed during life.

In 1862 Griesinger, in a paper on "Aneurisms of the Basilar Artery," details two cases of that disease, and refers to twenty-three other cases of aneurism of the different cerebral vessels, of which five had not previously been included in any enumeration.

In 1866 Lebert contributed a series of papers on aneurisms of the cerebral vessels, basing his observations upon eighty-five cases, all of which had, however, been previously published. In the same year a thesis was published at Paris by M. Gougenheim, in which he detailed more or less fully the particulars of twenty-five observations, and refers to twenty-five other published cases. Of the detailed observations one only, however, was an original case. The cases collected were made the basis of a full and able analysis of the pathology of these affections.

In 1868 M. Durand also published a thesis in which he extended the observations of M. Gougenheim by the addition of sixteen unpublished cases, of which nine are given in full. He appended to his collection reference to others, which had either been overlooked by M. Gougenheim or had appeared since the publication of his memoir, raising the total number of cases enumerated to 122. Of these cases he entered into an analysis having especial reference to the production of cerebral haemorrhage.

In the following year Dr. Hutchinson, in a paper in the second volume of the 'Pennsylvania Hospital Reports,' reported an original case, and tabulated the particulars of thirty-three other cases, of which, however, all but two or three had been previously collected together. Of these cases and fifty-one others collected from different sources he has given an analysis, having reference to the age and sex of the subjects, the seat and size of the aneurism, and the symptoms and results which they produced.

In 1872 the latest contribution to the pathology of the cerebral aneurisms was published by Dr. Bartholow, of Ohio, in the 'American Journal of Medical Science.' In this paper he describes in full an original case, and refers briefly to seventeen others which had been previously published, but of which six or seven had not before been collected. He then enters into an analysis of these cases, amounting in all to 114 cases,

and appends to them the results arrived at by Lebert and Durand.

In further illustration of the morbid condition under consideration I will now record the details of three cases of my own, and will supplement my paper by a tabular view of such of the published cases as can be arranged with reference to the following particulars:—The seat, form, and size of the aneurism; whether rupture had occurred; the state of the brain; the state of the other organs; and the history of the patient. The cases will be found classified according to the anatomical distribution of the internal carotid and basilar arteries and their branches.

**CASE 1.**—*Aneurism of the left middle cerebral artery; apoplexy; probably former fracture of the base of the skull (see Plate I, fig. 1, Plate II, figs. 3 and 4).*

James Ansell, æt. 34, admitted into Charity ward at noon on the 30th of October, 1875. About eight years before, while removing a truck in the street, he was struck by a cart and knocked down, pitching with the right side of the head on the pavement. When raised he was insensible, and blood was flowing from the right ear, nose, and mouth, and his eyes became bloodshot, but there was no external wound. He was taken to Guy's Hospital, and while there was put under chloroform, but probably only for examination. He remained in the hospital for about three weeks, and when discharged was apparently well; but soon after he got chilled, and was again admitted with an attack of rheumatic fever, for which he was under treatment for seventeen weeks. He then resumed his work, but three years after he had a second attack of rheumatism which lasted six weeks. He had ever since been subject to pains in the limbs after exposure to cold. He had latterly been employed as a labourer at the hospital. After the accident he was subject to pain in the head, and used frequently to place his hand on the right parietal region, and complain of pain in that situation. About five or six weeks before he had the last attack of illness, he was noticed by his friends to be getting more out of health. His appetite failed, and he was languid, sleepy, and depressed, and complained

more than usually of the headache, which he said extended to the forehead.

On the morning of the 30th of October, while at work, he was found sitting down in the corridor, and when spoken to by his fellow workman he said he felt very ill and had pain in his head, and thought he had had a fit. He was removed to the ward immediately, being admitted about noon. He was then listless and dull, and complained of headache; the tongue was dry and brown, and the breath offensive, the pulse was somewhat quickened, 104, and the temperature high, 102.9°. In the afternoon, at about 3 o'clock, he had a fit and became unconscious, and was still very torpid at five o'clock. His temperature had then risen to 103.2°, and the pulse was 112. He passed a good night, but at about nine o'clock on the following morning, or on the 31st, he was seized with convulsions. These partly subsided in a few minutes and then recurred with greater violence. At 9.30 the right pupil was much dilated, and the right side of the face was motionless, while the extremities, and especially those of the left side, were very rigid. At 9.45 he was again convulsed, the left side of the face and the left extremities being especially affected, and the left eyelid dropped. The ophthalmoscope did not display anything wrong with the eyes. Slight convulsive movements continued in the left arm and leg till 10.20, when he again had general convulsions. In a few minutes he became quite quiet, and lay with the limbs flaccid, the eyelids partly closed, and the right pupil contracted, the left dilated. The temperature at this time was 99°, the pulse 96, the respirations 10. He continued much in the same state till 1.45, when I saw him. He was then lying on his back with the limbs extended; the right arm and leg were relaxed and could be moved in any direction, while the left extremities were perfectly rigid. The head was drawn to the left side, and the muscles on that side of the neck were rigid. The right eye was open, the pupil very much contracted, and the globe turned to the inner canthus, and there was a constant twitching movement to the left, or in the opposite direction. The left eye was entirely covered by the lid, and when the lid was raised the globe was found much everted, and the pupil was greatly dilated and irregular in shape, the right side of the mouth dropped and was motionless,

the left side was slightly drawn up, and there was occasionally a twitching movement. He was breathing with a slight stertor, and was evidently comatose, so that it was impossible to arouse him at all, or to elicit the slightest sign of consciousness; the temperature was 101.4°. He continued in the same state till 9.30 in the evening, when he had a slight convulsive movement and immediately expired. His temperature at 3.25 p.m. was 100.8°, at 5.25 it was 101.4°, and at 8.40, 102.6°. After his first seizure the bowels were only slightly acted upon by an enema. He passed water in bed, but some was retained; it was quite free from albumen.

The body was examined twelve and a half hours after death. The lungs were slightly emphysematous, and the posterior parts somewhat congested. The pericardium was generally adherent to the heart, from which it was separated with difficulty. The heart was large, weighing 12 oz. Its substance was somewhat fatty, but the valves were natural. The liver was slightly fatty, and the spleen somewhat soft. The kidneys were slightly granular and contracted, the capsules adherent, and the vessels thickened.

On the removal of the dura mater a considerable quantity of blood was found effused beneath the arachnoid over the anterior portions of each hemisphere, especially in the parts corresponding to the middle fossa. There was also some effusion external to the arachnoid in the left temporal region and along both Sylvian fissures, but more abundant in the left. The arachnoid over the left parietal lobe was found to be adherent to the dura mater over the anterior and inferior part, and it was opaque at the vertex, and a sort of cavity containing yellowish semi-gelatinous fluid existed at the under surface of the left frontal lobe, and there was also an old softened patch at the base of the left parietal lobe over the fissure of Sylvius (Fig. 4). There was an extensive effusion of blood in the subarachnoidal space around the pons and medulla and extending down the spine. A rounded mass about the size of a small marble lay immediately at the outer side of the termination of the left internal carotid artery. This proved to be an aneurismal sac springing from the left middle cerebral artery close to its origin (Fig. 1). The sac was embedded in the temporo-sphenoidal lobe. It was filled with coagula, and had given way at one spot. The third nerve

where it passed beneath the tumour was somewhat flattened, of a yellowish colour, and softened. The middle cerebral artery at other parts of its course and the other vessels of the circle of Willis were natural, but the coats of the internal carotid and basilar arteries were thickened, and in places atheromatous. The right lateral ventricle contained some reddish fluid, and the left was filled with clot. There were two elongated openings leading into the tympanum on the middle of the upper part of the petrous portion of the temporal bone (Fig. 3), and the bone in front of these openings was raised above the level of that of the opposite side, and very thin. This may have been due either to original formation or the result of fracture.

In the compilation of the notes of the case during life I have been partly indebted to Dr. Turner, and for those of the post-mortem examination to Dr. Greenfield and Mr. Stevens.

This case is an interesting one not only as affording an example of rupture of an aneurism of one of the cerebral vessels giving rise to symptoms of apoplexy, but also as probably affording an instance of fracture of the base of the skull with recovery. There can be little doubt that the symptoms which, as described by his friends, attended the injury that he received eight years before death, were due to fracture of the base, and the fissures found in the petrous portion of the temporal bone were the remaining indications of such fracture. If this be correct the case is a very remarkable one. It is true that he ever after suffered at intervals from violent headaches; he was, however, able to follow his occupation till immediately before the fatal seizure.

The entire adhesions of the pericardium probably dated from the first attack of rheumatic fever which he had soon after the injury of the head, and the adhesions in the absence of any valvular disease must have given rise to the hypertrophy of the heart which existed. Cases of this kind show that, as would *à priori* be expected, simple adhesions of the pericardium do, by interfering with the free action of the heart, give rise to hypertrophy, though doubtless to a much less extent than valvular disease.

The condition of the brain and of the base of the skull in this case is figured in Plate II, figs. 3 and 4.

**CASE 2.—Aneurism of the right internal carotid artery immediately before it gave off the middle cerebral artery. Hemiplegia of the left side with partial impairment of intelligence, terminating in coma (Plate I, fig. 2).**

Elizabeth Chant, æt. 63, admitted into Alice ward, January 16th, 1877. As she had been living away from her friends in lodgings, it was not possible to obtain a satisfactory account of her previous state of health. All that was ascertained was that on the morning of the 12th she had complained of pain in the head and shoulders, and in the evening she was partially unconscious and paralysed in the left side. In this state she was admitted into the hospital on the 16th. When seen by myself on the 20th she lay in a very torpid state, but was capable of being aroused when spoken to in a loud voice, and she answered simple questions rationally. The left pupil was somewhat smaller than the right, and both were insensible to light. She could protrude her tongue, and the organ did not deviate, nor was there any obvious facial paralysis. Both eyelids drooped. The left arm was entirely powerless, but the left leg could be slightly drawn up. She had no difficulty in swallowing. Though two enemata had been exhibited the bowels had not been acted upon. She had passed water in bed. The sounds of the heart as heard at the apex were somewhat rough, giving the impression that with a more vigorous circulation there would be a murmur—presystolic or systolic. The pulse at the wrist was very feeble. She had no marked febrile disturbance. The temperature on the evening of her admission was only 98°, and afterwards it ranged between 98° and 99°. The urine was small in quantity, had a specific gravity of 1035, and did not contain any trace of albumen. She was directed to have the ammonia and bark mixture, and a blister was applied to the back of the neck. She continued much in the same state till the afternoon, when she was observed to have become more unconscious, and during the evening she became entirely comatose and breathed sterterously. The right pupil was much contracted, but there was no marked facial paralysis. She died at 2 a.m. the following day.

The post-mortem examination was performed by Dr. Greenfield twelve hours after death. On cutting through the dura

mater a thin layer of blood was found effused in the arachnoid over the anterior half of the right hemisphere, and over both posterior lobes there was also a thin intra-arachnoid effusion. Some effusion also extended upwards over the right hemisphere in the region of the Sylvian fissure, and to a much smaller extent over the left; and there was also some over the corpus callosum, and there was a large quantity of blood at the base. This effusion was traced to an aneurism about the size of a horse-bean, which arose from the right internal carotid artery immediately before it divides (Fig. 2). The aneurism extended along the course of the posterior communicating artery, but did not involve that vessel. The sac contained firm and partially decolorised coagulum mixed with fluid blood. The third nerve adhered to the sac, and was softened and of a reddish-yellow colour in that situation. In the right temporo-spheroidal lobe, where in contact with the aneurism, there was a cavity the size of a hazel-nut filled with blood and surrounded by softened and ecchymosed brain-tissue, and this communicated freely with the tip of the descending cornu of the lateral ventricle. There was slight softening of the posterior convolutions of the island of Reil, and the right crus and the optic tract also was softened probably from pressure. The optic chiasma and nerves appeared healthy. The brain was otherwise healthy, and weighed 46 oz. The vessels were entirely free from ordinary atheroma, but their size was small, and there was some irregular thickening in places. The longitudinal and both lateral sinuses were filled with firm coagulum, partly decolorised and partly retaining its natural colour. The veins on the surfaces of the hemispheres were also much engorged. The heart was about the natural size, weighing 9 oz. There was some thickening of the mitral valve and of the *cordæ tendineæ*. The aorta was large, but not atheromatous. The lungs were emphysematous, and the other organs healthy, except that the capsules of the kidneys were adherent.

In this instance the history is unfortunately defective, so that the symptoms recorded were only those observed while the patient was in the hospital and suffering from the fatal attack.

**CASE 3.—Aneurism of the right middle cerebral artery; atrophy and hemiplegia of the left side about a month before death; a second and rapidly fatal apoplectic attack.**

Mary Ann Clare, æt. 34, admitted into Alice Ward, St. Thomas's Hospital, under the care of Dr. Bristowe, on June 19th, 1873.

With the exception of an attack of rheumatic fever nine years before she had enjoyed good health till the commencement of the symptoms for which she was admitted.

About a month before that time she began to suffer from pains in the head, chiefly affecting the right side, and had noises in the head, and shortly after she had a fit in which she became unconscious. On recovering from this she was found to be paralyzed on the right side, and she remained aphasic for two or three days.

On admission there was complete motor paralysis of the left arm and leg, but the sensation was not impaired, and her power of speech was recovered. She was drowsy and stupid. The heart sounds were natural, and the urine free from albumen.

On the 20th of June she became suddenly unconscious, and rapidly died.

The body was examined by Dr. Payne the following day. The organs of the thorax and abdomen were healthy, with the exception of there being firm adhesions over the whole of the left side of the chest. The dura mater on the right side was tense, and on removing it a considerable mass of blood was seen under the arachnoid over the right anterior and middle lobes. In the right fissure of Sylvius there was a clot about two and a half inches in diameter, chiefly superficial, pressing upon the convolutions of the island of Reil, and then on both sides of the fissure. The anterior lobe was not deeply injured, but a clot also existed in the middle lobe, where it formed a diffused mass, easily separated from the surrounding brain substance. The clot only just touched the outside of the corpus striatum. It was mostly quite recent, but immediately around the artery of the Sylvian fossa there was some yellow pigment from old extravasation, and the brain substance around was similarly stained. There was also a little yellow pigment in the course of the Sylvian artery of the other side.

The brain was otherwise perfectly healthy. After washing the clot from the neighbourhood of the right Sylvian fissure so as to isolate the vessels, an aneurism about the size of a pea was found at the point of division of the vessel, and in the sac there was an aperture about the size of a pin's head.

For permission to publish this case I am indebted to Dr. Bristowe, and the notes are compiled from reports by Dr. Payne and Dr. Greenfield. In this instance the history is more satisfactory than in the last case, and it seems probable that the old blood-staining found about the Sylvian fissure and in the adjacent parts of the brain on both sides may indicate the occurrence of a partial rupture of the sac, giving rise to the attack of unconsciousness about a month before the last and rapidly fatal seizure.

The three cases which have been detailed correspond with the larger number of the similar cases that have been placed on record. In all of them the aneurism involved the internal carotid artery or its branches; in two, indeed, the sac was situated at the termination of that vessel immediately before it divided into its branches; and in the third it arose from the end of the middle cerebral trunk. In the majority of cases the aneurisms arise in these situations, though they are nearly as frequent in the basilar artery. In all the cases also the aneurisms became ruptured, so that the patient died from the large extravasation of blood which rapidly ensued. The sac was in two cases the size of a horse-bean, in the third of a pea—dimensions which have been largely exceeded in some of the recorded cases.

## Aneurisms of the Internal Carotid Artery.

Reference to author.	Seat, form, and size.	Result.	State of brain, &c.	State of the other organs.	History.
Dublin Journ. of Med. Sc., vol. i, Aug. to Nov., 1820, R. W. Smith, Case 4th, Dr. Mayne's case, P. 450	The aneurism was about the size of a small plum, and was situated at the termination of the internal carotid artery; it was completely filled with laminated coagulum of great density, buff colour and leathery consistence	The sac had given way	There was extensive extravasation in the outer and anterior part of the right hemisphere, and a coagulum in the brain which had burst into the lateral ventricle; the third nerve was amalgamated with the tumour, stretched and flattened, and had lost its natural colour; the optic was soft and flabby and of dingy colour	There was probably no cardiac disease	M., art. 33. In the South Union Hospital, 1860. Addicted to drinking, and after being very much overworked, and having had a debauch, he was found in the morning insensible, breathing stertorously, and with a congested face, cold surface, retention of urine, and inability to swallow. He recovered from this attack, and left the hospital, but continued listless, and his memory was impaired. He had also ptosis of right eyelid, an external squint, and impairment of vision of right eye. He continued in this state for four years, and he then had another apoplectic attack and died in a quarter of an hour.
Boudet, 'Sur les Hémorragies Méningées, Journ. des connaissances Médico-Chirurgicales, 1849,	The aneurism arose from the artery of the right side, just before the point at which it divides into its three branches; the sac was nearly separated from the vessel, there being an opening from it about three lines in diameter; the vessel in the seat of the aneurism was atheromatous, but elsewhere, though this state he was admitted into the hospital. The sensibility was impaired, especially on the left side. The eyes were closed, the pupils little sensible to light; the left nostril immovable, the right acting with the respiratory movements; the lips drawn to the right side; the left arm perfectly relaxed; the leg not diseased; the cavity contracted, but did not move when pinched; the right extremities were contracted; the face was pale, and the veins contained a clot	The sac was ruptured	The arachnoid, especially on the right side, was elevated by a large extravasation of blood extending through all the processes of the pia mater; the ventricles contained serum tinged with blood, and the vessels of the brain were distended	The pericardium contained a large bloody serum, and the cavities in the head and in the ears. These symptoms ceased, but recurred, and again ceased. One evening he had a violent emotion and passed a restless night. The following day he was taken ill while at work, and on going home fell as he was ascending the stairs, but recovered his intelligence in a few minutes, though he had headache and vomiting and was excited. The following morning he spoke sensibly, but complained of pain in all parts of the body, and during the day became unconscious and stertorous. In this state he was admitted into the hospital. The sensibility was impaired, especially on the left side. The eyes were closed, the pupils little sensible to light; the left nostril immovable, the right acting with the respiratory movements; the lips drawn to the right side; the left arm perfectly relaxed; the leg not diseased; the cavity contracted, but did not move when pinched; the right extremities were contracted; the face was pale, and the veins contained a clot	M., art. 57. In the service of M. Petit at Hotel Dieu. Two years and a half before, he had become deaf, and subject to pain in the head and ringing in the ears. These symptoms ceased, but recurred, and again ceased. One evening he had a violent emotion and passed a restless night. The following day he was taken ill while at work, and on going home fell as he was ascending the stairs, but recovered his intelligence in a few minutes, though he had headache and vomiting and was excited. The following morning he spoke sensibly, but complained of pain in all parts of the body, and during the day became unconscious and stertorous. In this state he was admitted into the hospital. The sensibility was impaired, especially on the left side. The eyes were closed, the pupils little sensible to light; the left nostril immovable, the right acting with the respiratory movements; the lips drawn to the right side; the left arm perfectly relaxed; the leg not diseased; the cavity contracted, but did not move when pinched; the right extremities were contracted; the face was pale, and the veins contained a clot

Reference to author.	Seat, form, and size.	Result.	State of brain, &c.	State of the other organs.	History.
Bullet de la Soc. Anat. 24e année, 1849, p.348. M. Barth's Lebert's Anat. Path. vol. i, p.575. Obs. cc	The tumour was situated behind the commissure on the left side, and compressed the motor left vessel, where it forms the posterior communicating artery, and was of a blackish colour; it may have been formed by dilatation of all the coats, and the internal tissues may have been destroyed; it was about the size of a filbert.	—	—	There were numerous small and suffered much from a winter cough for accumulations of some years. She had also ptosis of the left eyelid, and an external and upward squint of the eye. The pulmonary symptoms became aggravated, and the patient died in a few days with symptoms of subacute meningitis. The pupil was dilated and fixed.	F., sst. 70. She was a patient in Salpêtrière, in a state of profound coma, with contracted pupils and paralysis of the lower extremities, and died soon after. She had suffered from headache for two years, and a sense as if of the blows of a hammer over the right orbit. She lost the sight of that eye, and afterwards had difficulty in speaking and impairment of the sense of smell, and her intelligence failed. She slightly improved, but the paraplegia again commenced, and she had ensuing failure of power, suffered from palpitation, and became comatose.
Gaz. des Hopit., 1857, p.105. M. Giraudet.	The aneurism arose just at the point of origin of the right ophthalmic artery, and formed an oblong tumour on the right side of the sella turcica, 4 cm. long and 2½ wide; the sac was 2 to 3 mm. in thickness, its outer coat adhered to the adjacent parts, and the middle coat was thickened; it contained concentric cloths; the other arteries were healthy.—In this paper Feneil, Morgagni, and Burhove are said to	—	—	There was fluid in the sac of the arachnoid and in the ventricles; the anterior portion of the brain on the right side was excavated and softened, and the arachnoid and pia mater were wanting in this situation; the optic nerve was flattened like a ribbon, and the motor oculi and the ophthalmic branch of the fifth were also compressed; the cavernous sinus was obliterated	F., sst. 52. Admitted into hospital at Tours in a state of profound coma, with contracted pupils and paralysis of the lower extremities, and died soon after. She had suffered from

have seen bloody tumours in the brain, and cases are said to have been reported by Sandifur, Magendie, Ribes, Serres, Hodgson, Buschel, Albers, Nebel, Spurzyn, Copland, Kuring, and Krimer

Edinburgh Medical Journal, vol. iv, July, 1855, probably an aneurism of the left internal carotid to June, 1859, artery, close to the origin of the ophthalmic branch; the coats of the artery appeared spread over one side of the tumour, the other being formed of layers of coagulated fibrin and a week before blood; there was the remains of a cavity in the interior

Tumour size of spar-  
row's egg, placed beneath the anterior lobe of the cerebrum; it was a morœ, probably an aneurism of the left internal carotid to June, 1859, artery, close to the origin of the ophthalmic branch; the coats of the artery appeared spread over one side of the tumour, the other being formed of layers of coagulated fibrin and a week before blood; there was the remains of a cavity in the interior

The aneurism arose just before the giving off of the left ophthalmic artery; it was of small size and circumscribed; the ophthalmic artery was dilated, and had, together with the other arteries of the brain, earthy deposits in its coats

F., at 47. After having suffered for two or three weeks from loss of vision to a great extent, and for a week slight paralysis of the right side, was suddenly seized with unconsciousness, stertorous breathing, insensibility of pupils, and involuntary passage of evacuations, and died 47 hours after the attack.

F., at 65, whose case is described in 'Med.-Chir. Trans.' vol. xli, 1859, p. 175, as one of aneurism by anastomosis within the orbit. The carotid artery was tied, and after the operation she was paralysed on the same side and convulsed on the opposite side. She died sixteen days after the operation.

Brain above the aneurism was softened and disorganized

The aneurism pressed on the cavernous sinus, and there was some extravasation around it, probably from small rupture

Reference to author.	Seat, form, and size.	Result.	State of brain, &c.	State of the other organs.	History.
Ibid., vol. xii, 1860-61, p. 61.	Aneurism of that part of left internal carotid contained in cavernous sinus; it was of the size of a small nut, and contained laminated coagula on the outside and collection of cavity with fluid blood cases, cerily, within; it involved the p. 493; whole coats of the vessel, Brit. and which were elsewhere healthy; the third nerve For. Med. Chair. Rev., ran on the outside, and vo. xxxvi, was so thinned as to be p. 493; nearly divided; the St. George's fourth nerve was also Museum, much thinned, and the viii, 114 first division of the fifth was on the outside of the sac.	—	—	Patient under treatment for death with pain over the left eye and headache, followed by giddiness and ptosis of the lid, without history of rheumatism; pupil dilated and fixed, and the sight much impaired, and there was numbness of the cor- lymph on lining responding part of the forehead, but no headache or giddiness. He died with symptoms of heart auricle and on disease, dyspepsia, haemoptysis, &c.	Boy, <i>et. 16.</i> Taken about 2½ months before death with pain over the left eye and headache, followed by giddiness and ptosis of the lid, without history of rheumatism; pupil dilated and fixed, and the sight much impaired, and there was numbness of the cor- lymph on lining membrane of left membrane of left or giddiness. He died with symptoms of heart auricle and on disease, dyspepsia, haemoptysis, &c.
Gougenheim, Des Tumeurs Anérysmales des Artères des Cerveau, Paris, 1866, obs. xi, p. 56.	Sac at the end of the left carotid artery, and extended into the Sylvian artery; the sac had several projections, and its walls were atherosomatous.	—	—	Large clot covering the left frontal lobe, the heart hypertrophied, and the pia mater congested; at the base, in the Sylvian fissure, there was an older and firmer aorta extravasation about the size of a hazel nut	F., <i>et. 48.</i> Seized with apoplexy nine days before admission, and subject for some time to constant headache. When admitted she had hemiplegia of the right side, with partial in- patches in the hemiplegia, and she became worse and died in about two days.

<p>Path. Trans., vol. xix, 1867-68, p. 194. Dr. Bristowe, in Museum of St. Thomas's Hospital, No. 62.</p> <p>The aneurism arose from the left internal carotid at the point of division; it was spherical, flattened, and three quarters of an inch in diameter, and with two placed, and the projections the size of cherry stones.</p>	<p>The aneurism was embedded in the brain, under the membrane; and the optic nerve was flattened, and three quarters of an inch in diameter, and with two placed, and the lower portion of left an-</p> <p>factory obliterated; arteries and much diseased blood-stained and soft-ened</p>	<p>Extravasation in the arachnoidal sac and rom in aorta; health, nearly two months before death, with insensibility and paralysis of the right side. Patient partially recovered, but had inability to use the proper words. Was again seized with a succession of fits, and died the following day.</p>
<p>Lancet, 1869, vol. ii, of size of walnut, projecting into right cavernous sinus, and consisting of an aneurism of the internal carotid artery; it contained laminated, fibrinous deposits, external layers, firm and pale, the inner dark and soft.</p>	<p>Much fluid in ventricles, and brain softer than natural</p>	<p>M., at. 56. Right eyelid closed, partly from paralysis and partly from oedema, and surface red and superficially ulcerated, and choroid injected; surface of the eye insensible, and anaesthesia of right supra-orbital region of right side of nose and partly of right cheek. Six weeks before he had giddiness and pain in a limited spot in the right temporal region, and these symptoms were followed by the others. He died in about a month with albuminuria. Had primary syphilis in early life.</p>
<p>The aneurism arose from the artery of the right side; it was about the size of a horsebean, and contained firm and partly decolorised coagula; the vessels were irregularly thickened, but not atheromatous</p>	<p>Ruptured</p>	<p>After having had pain in head and shoulders for a few hours was taken with hemiplegia of the left side and partial loss of consciousness. Six days after became entirely comatose, and soon died.</p>
<p>Author</p>	<p>Heart of natural size; mitral ori-</p> <p>between the convolutions, fine rather small, the left side and valves some-what thickened and opaque; a-</p> <p>and orta rather large and adherent</p>	<p>Blood effused into the parietal lobe, be-</p> <p>and into the descending and valves some-what thickened</p> <p>ventricle; blood stain-</p> <p>of right optic tract and but not athero-</p> <p>matous; capillaries of kidney ad-</p>

## Aneurism of Internal Carotid Artery diagnosed eleven years before death.

Reference to author.	Seat, form, and size.	Result.	State of brain, &c.	State of the other organs.	History.
Trans. of Olin. Soc., vol. viii, 1875, p. 127, Jonathan Hutchinson.	The aneurism was the size of a bantam's egg, and arose from the artery of the left side, and occupied the inner part of the middle fossa of the skull; it was solid and the walls partly calcified, and had the shape of an egg with its Ophthalmic smaller end outwards, its larger resting on the bone; the internal carotid passed on its inner side, and there was an opening from the outer wall into the sac; the distal branches of the vessel were pervious; the cavity was apparently obliterated	—	The optic nerve was in close apposition to the tumour, but it did not appear to have been injuriously compressed by: the tumour rested on the Casserian ganglion, which appeared flattened, and the motor nerves of the eyeball were lost in the walls of the tumour which were partly calcified	—	F., <u>æt. 40</u> , was first seen in 1861, and then had marked ptosis of the left eyelid, with a slight convergent squint and inability to turn the eye outward. The pupil was fixed and dilated, and the power of accommodation was almost lost, these symptoms indicating complete paralysis of the sixth nerve and partial of the third, and probably of the ocular branches of vaso-motor also. The drooping of the eyelid had been first noticed a year before, with some dimness of sight. She had also at the time aggravation of severe headache, to which she was previously subject. There was throbbing in the <u>right</u> [this Mr. H. says was the record at the time, but he supposes it to be a mistake for <u>left</u> ] temple and under the ears, and some loss of sensation in the side of the forehead, and a bruit was heard. She died of aneurism of the abdominal aorta eleven years after. There was no proof of syphilis.
Lancet, 1875, vol. ii, pp. 489-874.	—	—	—	—	Lady, <u>æt. 40</u> , after suffering from influenza had severe neuralgia of right eyebrow, which, though constant, was subject to exacerbation. There was also some indistinctness of vision and diplopia, and two months after much photophobia, some dilation of pupil, and slight squinting. She also had a loud noise in the
Wm. E. Humble, of Corfe Castle	—	—	—	—	<i>Aneurism of Internal Carotid cured spontaneously.</i>

head, which was audible by those around her and through the stethoscope, especially in the right ear but not in the left. The operation or ligation of the carotid was proposed but declined. After a time she suddenly experienced a sense of something cracking in the eye, and the sound ceased, but there was still some headache, and the external squint was less. After five weeks had elapsed she entirely recovered.

*Aneurism of Internal Carotid Artery cured by operation.*

Association  
Medical  
Journal,  
1866, pp.  
1060, 1067  
account of  
case and  
discussion  
at Bath and  
Bristol  
branch of  
the  
Provincial  
Med. Assoc.,  
R. W. Coe

F., sc. 56. Five months before she was first seen she had received some blows on the head, and had exerted herself in lifting heavy weights, and immediately after had the sensation of a buzzing noise in the head; this was especially felt in the left ear and never after left her. She was also incapable of sleeping unless propped up in bed, and had frightful dreams and awoke in terror. No marked sounds were heard at the heart, but in the neck a systolic sound was heard, and also at the back of the head, and especially over the left petrous bone; when the carotid was pressed it ceased immediately, but after a time she still heard a slight

sound. There was a slight inward squint of the left eye, and she had a habit of winking with it, which occurred when first the buzzing was noticed. There was also some impairment of vision and slight diplopia. She could by effort abduct the eye as well as the right. The hearing was not affected. The anterior jugular vein was greatly distended. An aneurism of the internal carotid artery, immediately where it emerges from the petrous bone, was diagnosed. About a month after being first seen the left common carotid artery was tied. Immediately on the application of the ligature the rushing sound ceased, but she herself still heard it. For a time there was loss of power of abducting the eye. At first the dreams continued, but subsequently they ceased, and she slept comfortably, and recovered the power of abduction; and in about nine weeks she might be considered well, though there was still some indistinctness of vision.

## Arterio-venous Aneurism of Internal Carotid.

Reference to author.	Seat, form, and size.	Result.	State of brain, &c.	State of the other organs.	History.
Bullet de la Soc. Anat. de Paris, 30e année, p. 178, M. Henry	The internal carotid artery, where it crosses the cavernous sinus, was dilated and torn across so as to communicate with the sinus; the cavernous and sphenoidal sinus communicated freely; on the outer side a small flattened splinter projected into it, and lay against the motor oculi nerve; the ophthalmic vein was greatly dilated, but the artery was natural	—	At the posterior and internal part of the left side there was the cicatrix of a fracture, and around this the membranes were thickened and the brain inflamed, and the sinus projected	—	M., set. 21. Received a blow with an umbrella on the left eye, and the lower lid was cut. After this he had pain in the head and fever, but soon recovered, and the wound healed. The right eye, however, was prominent, and the ball was turned outwards and could not be depressed; the upper lid also drooped, and there was double vision. He was then seen by Vanguin, Sichel, Dumaine, and paralysis of the third nerve was diagnosed. He was then placed under the care of M. Nélaton. At that time the eye was much protruded, the lid dropped, and though the globe was fixed outward. There was distinct pulsation synchronous with the radial pulse, and a murmur was heard with the dilatation, and both these symptoms ceased when the carotid was compressed. There was also bleeding from the right nostril. Attempts were made to cure the disease by compression, but without effect, and the patient died, after having had repeated profuse bleeding, in about four months from the receipt of the blow. The symptoms were supposed to depend on an aneurism of the carotid or ophthalmic artery.

## Aneurism of the Anterior Cerebral (Artery of the Corpus Callosum).

Friedrich, Beiträge zur Lehre von den Groch-wilstein inner halb der Schädel. Halle, Wurz- enburg, 1858, p. 86	The sac had burst and the engorged and extravasated blood had escaped from the surface of the corpus callosum, and was of a rounded form and the size of a pea	Vessels of pia mater had sation on the surface especially at the base, and about the cerebellum and the fissure of Sylvius, and the course of the arteries on them; the course of the corpus callosum, and which had burst dilated; mitral valve perforated into the ventricles	Fluid in the pericardium; the valves of the aortic valve especially at the base, and about the cerebellum and the fissure of Sylvius, and the course of the arteries on them; the course of the corpus callosum, and which had burst dilated; mitral valve perforated into the ventricles
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anæmia of the coronary artery;  
anæmia thalæsia of a walnut in the splenic artery;  
spleen and liver enlarged;  
kidneys contracted

Lond. Med. Rep. Month. Journ. and Rev. by J. Copland, M.D., new ser., vol. iii, to Jan., 1825, nerves case by T. Spurgin, of Saffron Walden

The anæmia arose from the vessel of the right side, and was of the size of a hazel nut; it was situated on the outer side of the olfactory nerve, and before the junction of the optic nerves

The anæmia burst at its upper side into the lateral vein: it was engorged, and bloody fluid in ventricle with coagulum; very thick, and small vacuation in right hemisphere; coagula in base of adhesive tricle; softening of corpus striatum, and abscess in right anterior lobe of brain

Brit. & For. Med.-Chir. Rev., July to Oct., 1861, p. 494

The anæmia was situated on the vessel of the left side at its commencement; it was of the size of a three-penny piece, and was case cxixii; spherical, and had at St. George's fannated walls; it communicated freely with the artery, and contained soft coagulum

No. III. The papers of Dr. Ogle also contain the two cases published in the Path. Trans., together with those by Dr. Fuller and Mr. Holmes

Reference to author.	Seat, form, and size.	Bruit.	State of brain, &c.	State of the other organs.	History.
Arch. Gén. de Méd., 6e size of a pigeon's egg, 1868, t. xi, p. 229. M. A. Lerber, quoted from fibrinous concretions, a thesis, which nearly filled the case under the care of M.M. Levy, atheromatous patches; and Kuhn; the cerebral vessels appeared sound	The sac had ruptured, so were engorged with blood, and a large extravasation existed in the ventricles and at the base; there was an old apoplectic cyst, as large as a walnut, in the right anterior lobe of the brain at the level of the convolutions	—	The venous sinusses were engorged with blood, and a large extravasation existed in the ventricles and at the base; there was an old apoplectic cyst, as large as a walnut, in the right anterior lobe of the brain at the level of the convolutions	—	M., s <sup>t</sup> . 40. Taken in 1864 when in a cold bath (after bathing in the Rhine, Schützenberger) with a violent convulsive attack and loss of consciousness. Recovered intelligence in about ten days, but two days after had another attack of convulsions and coma followed by delirium. From this he again recovered and till now suffering from pain in the head and singing in the ears, but he was taken a third time with similar symptoms and rapidly died.
Pth. Tr., vol. x, 1858-59, p. 263	The aneurism was about the size of a horse head, and another about the size of a taro root. Dr. Bristow; in the left middle cerebral artery; the vessels near the base were thick in St.	The aneurism was about the size of a horse head, and another about the size of a taro root. Dr. Bristow; in the left middle cerebral artery; the vessels near the base were thick in St.	There was a large slit in the arachnoidal sac a few minutes and coagulated and especially at firm; the aneurism was at the thin part of the wall of the aneurism the base; clot also in contracted.	Heart healthy; heart atherosomatous and coagulated and especially at firm; the aneurism was at the thin part of the wall of the aneurism the base; clot also in contracted.	F., s <sup>t</sup> . 60, brought into the hospital insensible, and died in a few minutes. Had had a

### *Aneurism of the Anterior Communicating Artery.*

Thomas's Museum, No. 61	Med. Times and Gaz., vol. i, 1865, p. 357. T. W. W. Watson	The aneurism was about the size of a large horsebean. The other arteries were healthy	The sac had ruptured	There was effusion of blood beneath the dura mater	Tubercles in the lungs; heart and kidneys healthy	F., <i>est.</i> 49. Died suddenly in the night, but had suffered from pain in the head previously.
	Edin. Jour. of Med. Sci., vol. viii, July, 1862, Dr. John Struthers;	The aneurism was the size of a small pea, and the outer coat was con- tinuous with that of the artery; the other vessels near the aneurism were healthy, but the verte- bral, basilar and right carotid arteries had some slight patches	—	—	—	F., <i>est.</i> 80, said to have died of gradual decay, and not to have had any marked cerebral symptoms.
	See of Edin.	—	—	—	—	—
	MacDowell, Northum- berland County Asylum	The aneurism was situated immediately in front of the optic com- missure; it was of an oval form, $\frac{1}{2}$ in. in diameter, and $\frac{1}{4}$ in. in antero-posterior; it con- tained firm, dark clot adherent to the parie- tes; the walls had white patches	—	—	—	Aorta and coro- nary arteries a- theromatous; dilated; mitral valves thickened; liver large and fatty; kid- neys contracted

## Aneurism of the Anterior Communicating Artery.

Reference to author.	Seat, form, and size.	Result.	State of brain, &c.	State of the other organs.	History.
Path. Tr., vol. xvii, 1875-76, p. 2, Dr. Greenfield	Aneurism size of small pea on superior and anterior aspect of artery; the sac contained firm coagulated blood, and tery; it was surrounded by a layer of fibrous tissue, there had probably been a stated degree in the parietal lobes; there was also a large mass of coagulum, hemorrhages, as indicated at the base, and especially around the pons and medulla; there was congestion, firm, and blood-stained fluid, and very a small coagulum in the lateral and fourth ventricles	The sac had ruptured at its point of origin from the artery; it was surrounded by a layer of fibrous tissue, there had probably been a stated degree in the parietal lobes; there was also a large mass of coagulum, hemorrhages, as indicated at the base, and especially around the pons and medulla; there was congestion, firm, and blood-stained fluid, and very a small coagulum in the lateral and fourth ventricles	Blood was effused on the upper surface of both hemispheres, beneath the arachnoid, over the occipital lobes, that and in the Sylvian fissure, and to a less degree in the parietal lobes; there was also a large mass of coagulum at the base, and especially around the pons and medulla; there was congestion, firm, and blood-stained fluid, and very a small coagulum in the lateral and fourth ventricles	Slight atherosclerosis of the mitral valve; heart rather large, walls flabby, of eight days he had a fit, and soon died.	<b>M., ast. 45.</b> Fell from a scaffold at a height of thirteen feet. When admitted into St. Thomas's he was quite insensible, but after a time recovered his intelligence. At the end of eight days he had a fit, and soon died.
Bullet, de la Soc. Anat. de Paris, 7 année 1832, p. 26, M. Flandin	The aneurism arose at the point where the vessel of the right side divides. It was spherical, and had thick and firm parieties, and the cavity was filled with dark coagula	The sac was ruptured, and blood effused in the arachnoidal cavity, and there was very extensive softening of the right hemisphere, which seemed to have commenced at the base of the middle lobe near the fissure of Sylvius	M., ast. 60, admitted into the Hôtel Dieu paralysed on the left side, but with the intelligence entire. The attack had occurred three weeks before, and the patient died about six weeks after the seizure.		
		Much serum was effused in the arachnoidal cavity, and there was very extensive softening of the right hemisphere, which seemed to have commenced at the base of the middle lobe near the fissure of Sylvius	—		

## Aneurism of the Middle Cerebral or Sylvian Artery.

Bullet, de la Soc. Anat. de Paris, 7 année 1832, p. 26, M. Flandin

The aneurism arose at the point where the vessel of the right side divides. It was spherical, and had thick and firm parieties, and the cavity was filled with dark coagula

M., ast. 60, admitted into the Hôtel Dieu paralysed on the left side, but with the intelligence entire. The attack had occurred three weeks before, and the patient died about six weeks after the seizure.

—

The sac was ruptured, and blood effused in the arachnoidal cavity, and there was very extensive softening of the right hemisphere, which seemed to have commenced at the base of the middle lobe near the fissure of Sylvius

Prov. Med. Journ., 1861, p. 72, Dr. Fletcher Burn. Path. Soc.	Ruptured Aneurism of small size on the left artery	Extravasation on sur- face of left hemisphere and at the base, especi- ally on the left side. Small cavity in the an- terior lobe containing blood and communica- ting with the sac, and supposed to have been slowly forming without producing any marked symptoms	M., st. 38, who had suffered previously from lead poisoning was taken suddenly in the morning when watching a severe thunder storm, and supposed he had been struck. An hour and a half after this he was found prostrate, foaming at the mouth, and insensible, but soon partially recovered. Again became insensible and was convulsed, and lost the power of the right leg, and died nine and a half hours after this the first seizure.
Path. Tr., vol. vii, 1855-56, p. 125, Mr. Squire	An aneurism of the size of a horsebean arose from the artery of the right side, and was situated in the Sylvian fissure, and pro- jected into the anterior and middle lobes of the cerebrum; all the cere- bral arteries except the basilar and vertebrals were very atheromatous and rigid, and the in- ternal carotid and mid- dle cerebra especially so	Slight ha- morrhage, as if from thin- noidin places thickened; some subarachnoid effu- sion; old hemorrhagic carries in right corpus striatum and left hemi- sphere; brain substance around cranium not diseased	M., st. 57. Had an epileptic seizure six weeks and two months before death, followed by partial paralysis of left side and indistinctness of speech. A second attack of the same kind occurred soon after, but was partially recovered from. In twelve days had a third and rapidly fatal seizure.
Ibid., vol. xiii, 1861- 62, p. 2, Dr. Murchison	Ruptured to the extent of about a line	Blood extravasated about the aneurism in the fissure of Sylvius, but a large clot in the right hemisphere and hyperæmic in the ventricles, and about the pons and medulla oblongata; pia mater injected and with ecchymoses in places	M., st. 24, for some months subject to severe epileptic fits. Taken with apoplectic symptoms after drinking, and died in eight hours.

Reference to author.	Seat, form, and size.	Result.	State of brain, &c.	State of the other organs.	History.
Edin. Jour. of Med. Science, vol. viii, July, 1862, to June, 1863, p. 189. Dr. Haldane, and decolorised. The walls of portion of the sac were thick and atheromatous. There was no disease of the other vessels	Aneurism of branch of left middle cerebral artery. It was rather larger than a field bean, and the sac was partly filled with coagula, which were in part firm and decolorised. The	There was a laceration of the walls of the sac about an eighth of an inch in length	Thin layer of blood below the arachnoid and extending with the pia to the convolutions over the left hemisphere; a large extravasation into the substance of the hemisphere and the left corpus striatum; the cerebral substance otherwise free from disease	Blending of aortic valves, with thickening and vegetations; slight atheroma at commencement of aorta	Boy, ast. 14, patient in the Royal Infirmary under Dr. Warburton Begbie suffering from symptoms of obstructive aortic disease, and who was dull and heavy and complained of headache. He was taken suddenly with symptoms of apoplexy, but without decided paralysis, and died in a few hours.
Path. Tr., vol. xiv, 1862-63, p. 64. Mr. Callender for Dr. Burrows	The aneurism was situated on one of the chief branches of the left side. It was solid over the chief part of its extent, and lined by laminated coagula with a small cavity; at its base were three vessels, one of which was obliterated, while the other two opened into the sac; the walls of the sac were thick and hard, and cartilaginous	Rent in sac wall, and death from haemorrhage	—	—	M., ast. 68, subject for many years to severe epileptic seizures, but had none for eighty days, and his health seemed quite re-established. He was then suddenly seized with apoplexy and died in a short time.
Path. Tr., vol. xvii, 1865-66, p. 67. Dr. Broadbent	The aneurism arose from the right middle cerebral artery about half an inch from its origin. It was of small size, and was sessile on	There was a small rupture in the sac	Much blood was effused over the anterior lobes, especially on the right side and at the base, extending to the medulla oblongata	—	F., ast. 59, previously subject to vertigo, but otherwise in good health. Was taken suddenly with a fit, became comatose, and died the following day.

gata, along the fissure of Sylvius and into the third and fourth ventricles and hemispheres; the ventricles contained	There was bloody serum and clot under the membranes and in the ventricles, and upon the right side of the bulb and pons and between the lobes of the cerebellum	There was slight effusion of blood and serum in the arachnoidal sac and in the meshes of the pia mater below the left hemisphere. The sac was situated external to the origin and groove of the olfactory nerves between it and the island of Reil	Heart's walls very soft, and blood fluid; no disease of valves or of aorta; spleen large and soft; liver large and fatty; kidneys large, pale, and fatty
the artery. It was embedded in the anterior lobe of the brain	The aneurism was on a branch of the left Sylvian artery. It was the size of a large pea, and was situated near the point of the cornu Ammonis and upper part of the sphenoidal convolutions. There was no disease of the vessels	The aneurism arose from the artery of the left side, near its origin from the carotid. It lay in the fissure of Sylvius, partly sunk in the anterior lobe of the brain. It was the size of a small hen's egg, and contained partly fluid blood and partly firm fibrinous layers	Sinuses much congested, and the convolutions of the brain, especially on the right side, flattened. Large clot in the hemisphere, and the brain-substance around the optic thalamus broken down. The clot compressed, but had not entered the ventricle
Comptes Rendus et Mémoires de la Soc. de Biologie, année 1866, p. 85, M. Hayem	Path. Tr., vol. xix, 1867-68, p. 102, for Mr. Wood, of Parsons, of Bridge-water	Path. Tr., vol. xx, 1868-69, p. 111, Dr. Church, St. Barth. Hosp. Rep., vol. 1, 1869, p. 201	The aneurism arose from one of the branches of the left middle cerebral artery. It was the size of a small pea, and filled with recent blood. The cerebral arteries were free from atherosclerosis.

F., set. 21, in the service of M. Culmont at Lariboisière, taken suddenly six days after admission with dizziness and sickness, and fell down unconscious. She had nearly recovered on the third day, but the head was constantly drawn backwards. The coma then recurred, and she soon died.	F., set. 29. Three years before death had pains, regarded as neuralgic, in the left temple and side of face and nose, with dimness of vision. About seven weeks before fatal termination was suddenly seized with coma, which, however, soon subsided. The following week she had a second attack, and after it her intelligence was impaired and the pupils dilated and sluggish. In a few days she became paralysed on the right side. She again became better, but the paralysis remained, and she died suddenly in a fit.
	M., set. 40. Fall in the forest, and found quite insensible. Admitted into the hospital under Dr. Farre, and died in twelve hours. He was a distiller's man, and believed to be in good health up to the time of the seizure, but had been upped twenty years before for determination of blood to the head.

Reference to author.	Seat, form, and size.	Result.	State of brain, &c.	State of the other organs.	History.
Bullet. de la Soc. Anat. de Paris, xlviii, 1868; 2me ser., t. xiii, p. 599, M. Bourneville Path. Tr., vol. xx, 1868-69, p. 106, Dr. Bastian	Aneurism of the Sylvian artery. No atherosomatous disease of the vessels	—	—	—	F., 50. Was suddenly paralysed in the left arm and hand and face, but not in the leg, and without loss of sensation or of consciousness. Soon after her intelligence became impaired, and there was thickness of utterance and loss of memory of words. Two months and a half after, the paralysis of the left upper extremity remained, but there was no paralysis of the face, confusion of mind, or difficulty of articulation. About six weeks after she had a second attack with recurrence of the former symptoms. From this she again partially recovered, but four days before death she had a sudden comatose attack but without convulsions, and this continued till her death.
Bullet. de la Soc. Anat. de Paris, xlviii, 1868; 2me ser., t. xiii, p. 599, M. Bourneville Path. Tr., vol. xx, 1868-69, p. 106, Dr. Bastian	Aneurism of both middle cerebral arteries. That of the right artery was the size of a pea and of irregular shape, the artery leading to it being perivious. In the left middle cerebral artery the aneurism occupied the same position, and was of similar size, but it was occluded by firm clot, which was whitish and adherent. The arteries at the base were thick and opaque, but not notably diseased	—	The membranes of the brain were not materially diseased. There was evidence of old softening of the posterior part of the third frontal convolution close to the Sylvian fissure on the right side : a large aortic dark-coloured clot was found in the parietal and temporal lobes of the right hemisphere, and the brain was indurated. The left part of the corpus striatum and the neys	The mitral valve was thickened and adherent, and there was small vegetations on its edges and a half after, the paralysis of the left upper extremity remained, but there was no paralysis of the face, confusion of mind, or difficulty of articulation. About six weeks after she had a second attack with recurrence of the former symptoms. From this she again partially recovered, but four days before death she had a sudden comatose attack but without convulsions, and this continued till her death.	adjacent white substance was in a semi-diffuse condition ; there was also a smaller and older patch of softening, and in it a small aneurism in the anterior part of the left corpus striatum

The second convolution of the hemispheres, especially in the right side, was much flattened. The central portion of the right hemisphere was occupied by a large clot, and the ventricle was also filled

The convolutions of the hemispheres, especially in the right side, preserve much flattened vegetations, and the central portion of the right hemisphere was occupied by a large ot, and the ventricle was also filled

Boy, at 18, suddenly fell in the street, and was found quite insensible, and dragged his right leg, and in this state was taken to St. Bartholomew's. He vomited, and both left extremitieis were paralysed; he died in a few hours. About a month before he had been brought in a similar state to the hospital, but was quickly recovered under the influence of galvanism.

communicated with the artery, and was partly filled with clot. The larger sac had thin walls, apparently composed of pia mater, and opened from the former sac; it was filled with recent clot. The arteries at the base, including the middle cerebral up to the sac, were per-

no such hyper-trophied. The kidneys were intensely congested, and one of them had the remains of an infarct

rectly healthy. Tumor situated at point of reflexion of Sylvian fissure on the left side, cylindrical, 21 cm. long and 1½ wide. It was firm and contained yellowish masses; atheromatous; on the walls of the vessels at base not easily disengaged.

Much fluid under  
the cranioid and in spinal  
canal; pia mater thick  
and vessels engorged  
and colour of Sylvius  
and cerebral substance  
duscoloured. The tu-  
mour passed on the left  
side on the convolutions  
of the insula, which

The corpus striatum was partly destroyed. The optic thalamus was vascular and the optic thalamus was atrophied. The white substance of the right hemisphere was much congested. The anterior pyramidal body on left side was a grey colour, and smaller than the

F., set. 34. In the service of M. Delaunay at Salpêtrière, admitted in 1857, being then twenty-three years of age. When seventeen years of age she began to have attacks of hemiplegia of right side, and when twenty-two began to have aseptic symptoms followed by delirium. These continued to recur, and she died comatose after an attack in 1868.

Reference to author.	Seat, form, and size.	Result.	State of brain, &c.	State of the other organs.	History.
Durand, obs. iv, p. 29, M. Chorœus et Magnan	Small diverticulum in the left artery about 2 centimètres from its origin, and where it divides into its three branches. It would have held a lentil, and contained a resisting clot. The vessels at the base were very atheromatous and had thick plates on the walls, and the cavities were more or less narrowed, and this was especially the case with the left Sylvian and carotid arteries. The sac was apparently found only by the outer coat, the internal and middle being wanting. The outer coat was continuous with the artery, and had thick atheromatous plates in it	—	—	—	M., set. 86. In the service of M. Vulpius at Salpêtrière.
Durand, obs. vi, p. 32, M. Chorœus et Magnan	The tumour was situated at the origin of the artery on the right side. It was the size of a hazel nut, and did not contain any clot. The walls were very atheromatous and the sac was apparently formed by dilatation of the coats of the artery. The arteries at the base were very atheromatous	A collateral branch of the right Sylvian artery had burst	There was much exsanguination from the sac, and the sac ruptured.	There were general atherosclerotic changes in the brain, especially around the aortic valves and the Sylvian artery and its branches. There was a hemorrhagic mass in the white substance of the right hemisphere at the junction of the aorta; liver middle and posterior large; spleen firm; kidneys pale and granular	F., set. 60, admitted into the Salpêtrière in a state of imbecility, and with paralysis of the right side and albuminuria. Five weeks after admission she had an attack, in which she lost her consciousness and became paralysed on the left side, and shortly died.

<p>Thid., obs. iii, M. Lironville, p. 20</p> <p>Am. Jour. of Med. Sciences, N.S., vol. Ivi, 1869, Dr. W. D. Pepper, p. 403.</p> <p>Anat. xlii, 1<sup>re</sup> année 1869, 2me s<sup>e</sup> t. xiv, p. 492, M. Gazda, Brit. Med. Journ., vol. i, 1869, p. 74, Dr. E. Richard-Richardson</p>	<p>At the end of right carotid where vessel divides there was an aneurysmal sac the size of an acorn, with thick parietes, and partly filled with laminated clot. The arteries sclerosed and with atherosomatous plates</p> <p>The sac was occupied the left middle cerebral artery. It was about half an inch in diameter, and was situated just in advance of the corpus striatum, and to the third parietal lobe. The sac was filled with soft and firm dark clots. It involved all the coats of the vessel, with which it communicated by a small aperture</p> <p>The aneurism occupied the left artery and was the size of a hazel nut. It contained old clot, and the vessel displayed atherosomatous plates. There was a firm clot in the basilar artery from a branch of the left middle cerebral artery, and was as large as a horsebean</p>	<p>The sac pressed on the outer root of the right olfactory nerve, and the convolutions about the of heart; insula had nearly disappeared. The surface aorta; kidneys were depressed in that not materially diseased</p> <p>The sac was ruptured in the posterior sphere, on the outer side of the corpus striatum. There was no embolism of the corpus striatum, and to the lateral ventricles</p> <p>The aneurism occupied the left artery and was the size of a hazel nut. It contained old clot, and the vessel displayed atherosomatous plates. There was a firm clot in the basilar artery from a branch of the left middle cerebral artery, and was as large as a horsebean</p>	<p>Some opacity and thickening of the left valves and bronchitis, and died six days after admission into hospital. The left hand and fingers were contracted and atrophied.</p> <p>M., set. 48, with partial right hemiplegia, inability to utter articulate sounds, and loss of the memory afterwards. The paralysis became less, but he walked with an unsteady gait, and could not speak coherently and the aphasia continued. There was an aortic and mitral murmur. Three weeks after the lower extremities became edematous, and he died exhausted.</p> <p>F., set. 48, found in the streets insensible and with paralysis of the right side. The left pupil was dilated.</p> <p>F., set. 41, who had been imbecile for eight years. A year before her decease she had suffered from pain in the head, but it had ceased for some time. She was taken suddenly and soon died.</p>
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Reference to author.	Seat, form, and size.	Result.	State of brain, &c.	State of the other organs.	History.
Dublin Journ. of Med. Sc., vol. 1, Aug. to Nov., 1870, p. 446. R. W. Smith, Case 2	The left middle cerebral artery was obstructed by a plug of fibrin of a yellowish colour for fully an inch, and where the obstruction existed it was dilated so as to form an oblong tumour half an inch long and a quarter of an inch broad; elsewhere the artery was smaller than its fellow, and the other arteries of the circle of Willis beyond the tumour	—	Brain displayed a softened space in the middle lobe	Pericardium adherent, and thickening and vegetations of the aortic valves	F., at 26, admitted into Whitworth Hospital under Dr. MacDowell in 1865. Had been under treatment for rheumatic fever two years before, and then had symptoms of aortic incompetency. A month after admission she had an attack of bronchitis, and after about another month she became hemiplegic in the right side, and her intelligence became much impaired. She got bedsores, and gradually sank.
Echeverria on Epilepsy, New York, 1870, Case 1, p. 53	Dilatation of the artery of the left side, at the point where it gives off the anterior branch. The artery was occluded, and the aneurism apparently originated in embolism	—	There was a small tumour of the dura mater. The arachnoid was filled with coagulated blood. The convolutions were flattened, and the heart was enlarged	There were dura vegetations in the aortic and mitral valves, the groin, and an echarotic under the right side of the chin. She was a patient in the Hospital for Epilepsy.	F., at 12, subject to fits and paralysed on the right side of face and in the left limb, especially the arm. There was a cicatrix in the right side of the chin. She became soporose, and died in a fit.
Ibid., Case 6, p. 72	The artery of the right side was occluded by a firm, laminated, orange-coloured clot, which adhered to the walls. The vessel was dilated	—	The arachnoid was opaque at the anterior part of the base of the brain, and there was serous effusion beneath it. Film of congealed blood over large portion of anterior and middle lobes of brain, and yellow granulations on choroid plexus, and patches in centrum ovale	Heart natural, lungs tuberculous	F., at 26, suffering from constitutional syphilis, with obstinate headache and epileptic convulsions, palsy of left eyelid, and paralysis of left limb, and gradual failure of mental power. She became soporose, and died in a fit.

Path. Tr., vol. xxiii, 1871-72. The sac arose from the left artery, and was burst by a very large pea: it lay in the extra-Sylvian fissure. The arteries at the base were highly atheromatous

Path. Tr., vol. xxiii, 1871-72. The sac had burst by a very small opening, about the size of a very large pea: it lay in the extra-Sylvian fissure. The arteries at the base were most abundant there, about the optic commissure, but was not missive, and to a less at commencement of aorta.

F., at. 72 to 74, died quite suddenly while at dinner in a friend's house, where she had just arrived. She was supposed to be in good health previously.

An aneurism the size of a pea was found at the point of division of the history of the right Sylvian artery a pin's head the case was reported by Dr. Greenfield and the post-mortem examination was made by Dr. Payne. Case 3 of this paper, p. 135

The sac had burst at an aperture in the size of a pin's head at the anterior point of division of the right Sylvian artery a pin's head the case was reported by Dr. Greenfield and the post-mortem examination was made by Dr. Payne. Case 3 of this paper, p. 135

There was slight effusion of blood into the arachnoidal cavity at mitral and aortic valves, and some more abundant in the constriction of Sylvian fissure and the mitral orifice: atheroma was not missive, and to a less at commencement of aorta. Right kidney and medulla, and between the medulla and cerebellum. The ventricles and spinal canal were free from blood, and the convolutions not flattened

There was slight effusion of blood into the arachnoidal cavity at mitral and aortic valves, and some more abundant in the constriction of Sylvian fissure and the mitral orifice: atheroma was not missive, and to a less at commencement of aorta. Right kidney and medulla, and between the medulla and cerebellum. The ventricles and spinal canal were free from blood, and the convolutions not flattened

Much recent blood was effused under the healthy, the liver large, and the kidneys slightly enlarged. The heart was of the right fissure of Sylvius, pressing upon the convolutions of the island of Reil and on each side, and involving the substance of the brain on each side and at the anterior lobe; around the fissure was some yellow pigment as if from old extravasation, and there was a similar condition in the neighbourhood of the right Sylvian artery

F., at. 34, under the care of Dr. Brittoe at St. Thomas's Hospital in June, 1873. Seven years before she had had rheumatic fever, and a month before she had begun to suffer from pain in the head. Shortly after she had an attack in which she became unconscious, and which left her paralysed on the right side and aphasic for two or three days. A week after she had a second similar attack, after which she was paralysed on the left side. When admitted she had complete loss of motor power on the left side, and she was drowsy and stupid, but her speech was unaffected. Three days after she had a third seizure, and died comatose in about an hour.

Reference to author.	Seat, form, and size.	Result.	State of brain, &c.	State of the other organs.	History.
Archiv Gén. de Méd., 1875, vol. ii, vise aneurism about the size of a small cherry, and it was perforated, p. 728, extracted in the Revue Clinique Médicale. In the service of Prof. Westphal.	The principal anterior branch of right Sylvian artery; there was an aneurism about the size of a small cherry, and it was perforated	Ruptured	In the right temporal region there was a clot the size of a fowl's egg; and in the frontal region one as large as a nut, surrounded by softened and discoloured brain tissue; clots also about the chiasma and effusion in the spinal canal	—	M., st. 46, when seen was somnolent, eyes closed, congested face, carried at times hand to his head and moaned; could be aroused to answer questions; rigidity of neck and difficulty and pain in raising the head; no signs of paralysis, and pupils natural; hypersensitivity of surface of neck. Twenty days after the rigidity and hyperesthesia became less, and he was more intelligent. In the evening he became worse, was delirious; three days after had severe pain in the head, and two days after became comatose and the left extremities were paralysed, and he died in about six days.
See p. 129 of the paper.	The aneurism formed a rounded mass, situated on the outer side of the left internal carotid artery at its termination, and across the middle cerebral artery immediately at its origin. The arteries at the base were otherwise healthy	The sac had burst at its upper part	There were remains of a fracture of the petrous portion of the temporal bone. Recent but was easily removed; heart was easily removed; heart after subject to headache and pain in the limbs. For five or six weeks before his fatal seizure he was failing in health, and suffered more severely from headache. He had a fit while at work, and was found insensible. He partially recovered his intelligence, but the following day had another convulsive attack and became comatose, and died about two days after the first seizure.	The pericardium was generally adherent, but valves natural; liver slightly enlarged; spleen over the pons and at soft; kidneys the inner side of the slightly granular left middle cerebral and contracted fissure. There were softened patches on the under surface at the left anterior lobe and in the Sylvian fissure. There were	M., st. 34. Eight years before had been knocked down by a cart in the street and sustained very severe injury of the head, but without external wound. Very soon after he had rheumatic fever, and was ever after subject to headache and pain in the limbs. For five or six weeks before his fatal seizure he was failing in health, and suffered more severely from headache. He had a fit while at work, and was found insensible. He partially recovered his intelligence, but the following day had another convulsive attack and became comatose, and died about two days after the first seizure.

## Aneurism of Vertebral Arteries.

Schmidt's Jahrb., 1866, 130 Bd., p. 161; quoted from Aguise, Bd. xxvii, No. 9, 1866	Aneurism of the left vertebral artery forming a rounded bluish- coloured tumour. The tumour lay in front and to the right side of the medulla oblongata	Dura mater adherent to the right lung. She had previously had diffi- culty in speaking, and threatened paralysis of the lower extremities.	F., sit. 61. Died in 1864 of inflammation of the right lung. She had previously had diffi- culty in speaking, and threatened paralysis of the lower extremities.
Bullet. de la Soc. Anat. de Paris, 1869, année 2me série, t. xiv, M. Lorne	Aneurism of left ver- tebral artery	Ruptured	Extravasation in the meninges
Echeverria on Epilepsy, New York, 1870, Case 24, p. 124	Dilatation of both vertebral arteries, with atheroma of the coats and plugging of the cavities	The right artery near its junction with the left to spinal canal. Spinal accessory never formed the basis upon, and the brain was softened, especially the two corpora striata	Blood was extrav- asated about the pons and extended into the spinal canal. Spinal accessory never pressed upon, and the brain was softened, especially the two corpora striata
M., sit. 19	—	—	M., sit. 67, subject to epilepsy from early life. Four months before death suffered from dizziness, and had difficulty in protruding the tongue and in swallowing. The fits decreased in frequency, but the loss of power of move- ment became greater, and he had cramps and pains in the limbs and numbness in the feet, and difficulty in articulation. The intelligence was only occasionally slightly impaired.

Reference to author.	Seat, form, and size.	Result.	State of brain, &c.	State of the other organs.	History.
Bullet de la Société Anatomique de Paris, xviii <sup>e</sup> année 1872, 2me série, t. xvii, p. 415, M. Sevestre	There were two aneurisms on the left vertebral artery, and one of these, about the size of a pea, was ruptured; the other aneurism was only a small coronary dilatation. The left vertebral artery was altogether larger than the right, and it was slightly atheromatous. All the other vessels were quite healthy	—	There was subarachnoid extravasation extending from the brain to the spine. It covered the inferior surface of the brain, but was less at the sides, and only in places above. The portion of brain beneath the minute ventricle was destroyed, and there was blood in the fourth ventricle, but not in the lateral ventricles. The extravasation extended down the spine to the cauda equina, and was especially marked in the dorsal and more particularly the lumbar regions on the posterior aspect	Viaecra of the thorax and abdomen sound, on going to bed at night, with what seemed slight to have consisted in a succession of shocks of which left him generally depressed and with intense headache, and in the morning attempting to rise found his limbs weak. He had never previously had anything of the kind. Two days after he was able to return to work, having only some remains of headache and weakness in the lower extremities. Ten days after he had another attack without obvious cause, but of more severity, and in which he lost consciousness, and when he recovered after an hour he could only move his legs with difficulty, and had great pain in the head and loins. He was then taken to the hospital. When seen three days after he had violent pain over the whole head, but especially in the occipital region, but the lumbar pain had nearly abated. He had some paresis of the lower extremities, but no paralysis of the face and upper extremities, and no loss of sensation anywhere. The following day he had another convulsive attack with coma, and similar attacks recurred at intervals. The loss of power in the lower extremities continued, and he had retention of urine. Though he denied having been intemperate it was probable that he was so. This illness, it was thought, might be due to syphilitic disease of brain. He died in about ten days after his admission.	M., est. 45, in Hôpital Lariboisière, under M. Jacquot. Fifteen days before he was taken, on going to bed at night, with what seemed slight to have consisted in a succession of shocks of which left him generally depressed and with intense headache, and in the morning attempting to rise found his limbs weak. He had never previously had anything of the kind. Two days after he was able to return to work, having only some remains of headache and weakness in the lower extremities. Ten days after he had another attack without obvious cause, but of more severity, and in which he lost consciousness, and when he recovered after an hour he could only move his legs with difficulty, and had great pain in the head and loins. He was then taken to the hospital. When seen three days after he had violent pain over the whole head, but especially in the occipital region, but the lumbar pain had nearly abated. He had some paresis of the lower extremities, but no paralysis of the face and upper extremities, and no loss of sensation anywhere. The following day he had another convulsive attack with coma, and similar attacks recurred at intervals. The loss of power in the lower extremities continued, and he had retention of urine. Though he denied having been intemperate it was probable that he was so. This illness, it was thought, might be due to syphilitic disease of brain. He died in about ten days after his admission.

Prov. Jour., vol. i for 1872.	The left vertebral artery in its fourth stage displayed an aneu- rial dilatation. The vessels were diseased	The seat of rupture is not lum over the pons and peduncular space, and the course of the fi- bres of Sylvius, espe- cially on the left side. No extravasation in any part of the brain.
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M., st. 60, who had delirium tremens. Taken in the morning with symptoms of apoplexy. Admitted into the Meath Hospital with symptoms of coma, and died in twenty-four hours after seizure.

### Aneurism of *Basilar Artery*.

Bullet. de la Soc. Anat. de Paris, 3me série, t. 2, 2me anné 1836, p. 22. M. Lebert	Aneurism of the size and form of a hen's egg (21 lines long and 15 wide). It was firm, mammillated, and with yellow plates on its sur- face. It compressed the bulb and cerebellar lobes. The three coats of the artery could be traced into the sac, but not to the summit. It was nearly filled with a dense, grey-coloured, fibrinous mass.	There was no marked effusion under the a- rachnoid or in the ven- tricles. The upper part of spinal cord had a layer of a yellowish colour, and was soft- ened. The roots of the hypoglossal nerves were destroyed, and the pneumogastric and glossopharyngeal were flattened. The annular protuberance was only about one fifth of its proper size. The pyra- midal bodies were de- stroyed, and the olfactory bodies pushed aside	M., st. 68. Taken suddenly seventeen years before death with loss of power over the lower limbs. Partially recovered, but after- wards became gradually paralyzed both in the upper and lower extremities. The intellect became weak, he had sense of weight and pain in the head, and occasional attacks of dizziness and sense of suffocation and dyspnoea. He had also dysphagia, and almost entire loss of voice. He died in an attack of dyspnoea.
Engel. Cesterr. Med. Jahh., 1841, série Griesinger, obs. 22, p. 564	A double sac on the basilar artery; the an- terior portion as large as a plum, the posterior as a hazel nut, contain- ing firm, fibrinous co- agulum	The sac had burst.	F., st. 17, who died suddenly.
		The sac extended in front of the pyramidal bodies to the anterior portion of the pons.	The whole right side of the medulla ob- longata and pons was much atrophied. The fifth and tenth nerves of the right side were reduced to a few filaments

Reference to author.	Seat, form, and size.	Result.	State of brain, &c.	State of the other organs.	History.
Topati, Bullet. de Biogna, Loglio, 1844; Schmidt's Jahrb., bd. 46, 1846, p. 265, Griseinger, p. 561, obs. 12 Lond. Med. Gaz., vol. xxviii, 1846, N.S., vol. iii, p. 384; Dr. Eager in Report of Manchester Path. Soc.	Basilar artery dilated for an inch in length and half an inch in width, and filled with coagulum	—	—	In the parts around medulla oblongata there was a marked compression.	M., set. 40. For ten months before death he was subject to dizziness, tottering gait, weakness of limbs, and general paralytic.
					M., set. 58, began to complain nine months before death of pain in the head, extending from the back to the front. Three weeks afterwards had paralysis of the right portion of the face, but without any deviation of the tongue or impairment of the power of deglutition. Two weeks before death became hemiplegic on the left side, with difficulty in pronouncing the tongue and in swallowing, sometimes so great as to threaten suffocation. During his whole illness there was no loss of memory or mental power. He died suddenly in a fit of laughter.
					M., set. 33, admitted into New York Hospital in 1848, having had slight attack of apoplexy six months before. After this had loss of power of smell and inability to recall the proper names of things, and impaired power over the lower extremities, and dilated and partly insensible pupils, but no headache. About six weeks after had another attack of coma and convulsions, and died.

M., st. 54. Loss of sensation and motion on right side. Died six weeks after coming under observation.

The basilar artery in its anterior portion, and the two deep cerebral arteries were dilated in places, and especially towards the lower end were thickened and widened.

Ulrich, Deutache Klin., 1856, sac filled with coagulum and the circulation through the artery altogether arrested Lebert, Anat. Path., Paris, 1857, part of the basilar artery. It was about the size of a small olive vol. i, p. 574, and pl. xxxi, fig. 4

The aneurism produced a slight depression of the annular protuberance, but with this exception the brain was healthy, and the motor oculi not compressed

M., st. 55. Loss of sensation and motion over the diseased parts of the artery was softened, and contained a cyst in the interior. There was also a cyst in the left thalamus

M., st. 85. Several times attacked by syphilis. Taken with weight in the head and pleurisy, and became apoplectic, and was paralysed.

F., st. 70. Under M. Valjeau in La Charité with fracture of the neck of the thigh bone. Had been paralytic on the left side for about ten years, but had partially recovered. She never had the intelligence impaired or lost; her sight or the power of hearing, and could speak well. The sensation was not lost. Died of pleurisy about five months after admission.

F., st. 24. After endocarditis had rotation and inwards of left eye, and diplopia two months there were infarcts in the tracted, and there was general paralysis.

The heart was diseased, and the spleen and kidneys, and the splenic artery was obstructed

The basilar artery communicating and posterior cerebellar arteries. The basilar artery about the left crura cerebri and near the posterior cerebral and the motor oculi was dilated, and there was an extravasation of blood in the adjacent part

Oppolzer, Wien. Med. Woehnsch. 1860, p. 132, Grieninger, p. 659, obs. 4

Reference to author.	Seat, form, and size.	Result.	State of brain, &c.	State of the other organs.	History.
Bullet, de la Soc. Anat., xxv, année 1860, 2e série, t. v, p. 321; Gueniot; case also reported upon by M. Maillardin	The left vertebral artery and basilar both much dilated, the former being of the size of a very large goose-quill, the latter of an ordinary one. There were plates in the coats, and the internal coat was separated from the two other. The other arteries not diseased.	Dilated vessels not apparently ruptured.	The membranes and exterior of the brain natural, but there was an extravasation occupying nearly the whole of the interior of the protuberance. The coagulum was the size of an almond, and was imperfectly separated into two portions	—	F., at 47, formerly of intemperate habits, at Hôpital St. Antoine in the service of M. Boncher in 1859. For sixteen years subject to hysterical attacks with pain on the top of the head. Three days before admission taken with weight in the head, vertigo, and singing in the ears, and soon after became paralysed on the left side and could not speak, but did not lose consciousness or the sensation in the paralysed parts, and died in three days.
Archiv der Heilkunde, Leipzig, 1862, Griseinger, Case 1, p. 548, and Case 1, p. 550, obs. 5	The basilar artery for the anterior half of its extent was hard and irregularly dilated, and its cavity filled by firm, yellowish-grey fibrinous coagula, which were laminated and adherent to the coats. In the sora was a cavity about the size of a hemp-seed, containing soft coagulated blood. The other arteries were atherosomatous.	—	The processus atlantum and the pedunculus cerebri were superficially softened	The valves of the heart were healthy, but the muscular substance attack was taken during the night with paralysis of motion and sensation in the left arm and the leg, especially the former. After about five weeks she entered the hospital, and five days after became comatose during the night, and died the following morning.	F., at 48, had suffered for three or four years from dizziness and singing in the ears, and after having had a more severe attack was taken during the night with paralysis of motion and sensation in the left arm and the leg, especially the former. After about five weeks she entered the hospital, and five days after became comatose during the night, and died the following morning.
Ibid., Case 2, p. 553, obs. 20	The left vertebral and basilar arteries were atherosomatous and much dilated	—	—	—	M., at 56, became asthmatic in 1857, and in 1860 had much dry cough, but without beating of the heart or oedema. He entered the hospital, and died in about a week.

Bullet. de la Soc. Anat., xxviii, 1862, goose-quill. The coats from it were thickened and increased, p. 385, filtrated with a whitish, M. Blache's granular, fibrinous material, and the sac was filled with decolorised clot. Except at the seat of disease the coats of the vessel were healthy, and the clot did not involve the other vessels

The artery in its middle and towards its termination was dilated to the size of a large clot. The coats from it were thickened and in-

M., st. 42. An officer in an infantry regiment who had repeatedly had syphilis and giddiness with loss of consciousness. Two days before admission into hospital he had a more severe attack, in which he was insensible for half an hour, but was not convulsed. His intelligence was impaired; he spoke slowly and had a tottering gait. He complained of constant weight and pain in the head, but there was no paralytic symptom. Soon after he fell down unconscious, and died in five minutes.

A layer of recent blood covered the base and small aperture of the brain under the arachnoid, and between the membrane and atheromatous pia mater. Its sides ended cicatrix in the greater lobe of the bulb, entered the fourth ventricle, sent a small projection into the middle ventricle, and extended into the spinal canal. It was thickest about the bulb. The brain was somewhat softened at the lower and middle part of the right side, but there was no clot in the lateral ventricles. eight years before

The sac was

The aneurism was about the size of a pea. The other portions of the vessel and the other cerebral arteries were only slightly atheromatous

Path. Tr., vol. xvi, 1864-65, p. 88, Dr. H. W. Fuller; Dr. Ogle's cases, Brit. and For. Med. Chir. Rev., vol. xxvi, p. 493

The heart was soft and flabby, and the valves of the heart under the aorta were for a year he had been subject to attacks of giddiness with loss of consciousness. Two days before admission into hospital he had a more severe attack, in which he was insensible for half an hour, but was not convulsed. His intelligence was impaired; he spoke slowly and had a tottering gait. He complained of constant weight and pain in the head, but there was no paralytic symptom. Soon after he fell down unconscious, and died in five minutes.

M., st. 87. He had received a blow on the head when in the Crimea, and ever afterwards was greatly excited by stimulants, even by taking a single glass of beer. He did not, however, suffer from headache, and otherwise enjoyed good health. Five days before admission into St. George's he had severe diarrhoea, and the following day had five epileptic fits in rapid succession, and did not recover his consciousness after them. He then recovered, and appeared tolerably well, and did not complain of headache, but the evening before his admission he had another severe fit followed by coma. While in the hospital he lay still, but was only partially conscious, and was not paralysed. The day after his admission he had a very slight convulsive attack, and died in a few minutes.

The heart was healthy, but the aorta was slightly atheromatous. One kidney contained a large cyst, but otherwise the vessels unusually constricted. There was a large clot in the ventricles, and extravasated blood also at the base, the oblongata being completely embedded in it. The clot extended down the spinal canal to the canalis equina

Reference to author.	Seat, form, and size.	Result.	State of brain, &c.	State of the other organs.	History.
Archiv der Heilkunde, Leipzig, 1865, Dr. J. Varenntrapp, p. 85, Aneurisms. Arteria Basilaris.	In front of the pons there was an aneurism of the basilar artery the size of a large pea. It consisted of a limited dilatation of the artery. The arteries were not diseased either in the seat of the aneurism or elsewhere	There was a rupture in the sac	Fluid was effused in the cavity of the ventricles and in the membranes. There was an extravasation at the base, especially about the pons, which extended also into the substance of the brain and the ventricles	The heart was slightly enlarged, and the mitral valve thickened and both carthagenous and stenosed, and the left ventricle considerably hypertrophied. The liver was in the coming stage of cirrhosis. The right kidney was fatty, and the left had a greyish-coloured infarct	F., est. 51, in the Heilengesicht Hospital in Frankfurt. Taken suddenly with pain in the back of the head followed by loss of consciousness, which was, however, soon moved. The pain in the head continued, and there was singing in the ears with delirium and stupor, and she died in about ten days.
Bouchard, Etude sur quelques points de la Pathologie des hémorragies Cérébrales, Paris, 1867, p. 64.	The basilar artery on its left side was dilated to the size of a haircot bout the size of a bean and toruous. The left Sylvian artery was also dilated at its origin. Two milky aneurisms. The cerebral vessels were generally thickened and atheromatous.	A military aneurism	A tumour of considerable size was found in the left parietal lobe formed by two large veins with a pachycephalous patch near them. The aneurism of the basilar artery had produced a depression in the protuberance.	A tumor of the brain substance around the clot was softened. In the right corpus striatum and thalamus there were old cysts	F., est. 61, under M. Charcot. Long paroxysm on the left side. She could not walk, her intelligence was much impaired, and her speech very defective. She had an attack of an apoplectic character which left paralysis of the right side, and she died comatosed in two days. The lower extremities were edematous.

<p>Path. Tr., vol. xx, 1868-69, p. 112, Dr. Sample</p> <p>The aneurism arose at the points of division of the artery into its superior cerebellum and posterior cerebral branches. It was quite central in position, and was of the size of a small bean. It is suggestive that the aneurism in right had originated in embolism</p>	<p>The aneu- rism had burst at the points of division of the artery into its superior cerebellum and posterior cerebral branches. It was quite central in position, and was of the size of a small bean. It is suggestive that the aneurism in right had originated in embolism</p>	<p>There were considerable adhesions between the base of the brain between the anterior part of the pons and the optic commissure and the aortic valves along the fissures of Sylvius, but there was no blood in the ventricles</p>	<p>F., ast. 36, suddenly seized while under treatment for an attack of bronchitis with pain in the head and immediately became insen- sible. She partially recovered her con- scientiousness, but the coma again occurred, and she died in twenty days. At one time the urine was retained and then there was some albumen in it, but it was at other times free from albumen. There was a slight ecchy- mosis over the right eye from an accidental blow received a month before.</p>
<p>Durand, Des Auér., 1868, p. 46; case p. 48; also, reported by Bouchard, Pathologie des Hémor- rhagies Cérébrales, Paris, 1867, obs. ii, p. 34</p>	<p>The basilar artery was dilated to three or four times its natural size. It had hard plates in its walls, and was tortuous, obs. viii, and the vessels of the pia mater contained a fibrous thrombus. The arteries generally were atheromatous</p>	<p>There was a large extravasation of blood in the arachnoidal sac, which communicated with an extensive clot in the left posterior lobe, and the cerebral substance around the atheromatous; was softened. The other organs not examined</p>	<p>The heart of natural size; valves indurated, but no alteration was injured by a carriage. Two months before her admission she was found in bed unconscious, and she was left fatuous and paralysed and with the arms contracted, especially in the right side, but without marked loss of sensation. Four months after admission she had another convulsive attack followed by coma and stupor, and soon died.</p>
<p>Ibid., obs. ix, p. 52, case of M. Bamberg, Wurzburg</p>	<p>An aneurism of the basilar artery of the size of a cherry, the vessels otherwise healthy</p>	<p>There was an extravasation of blood at the base of the brain</p>	<p>There was an opening in the sac, the size of a pin's head</p>
		<p>Digitized by Google</p>	<p>Heart and other organs presented nothing unusual</p>

Reference to author.	Seat, form, and size.	Result.	State of brain, &c.	State of the other organs.	History.
Bullet. de la Soc. Anat., xiv, Année 1869, with the 2me série, t. xiv, p. 188, M. Berger	The aneurism arose at the point where the left vertebral unitas with the basilar. It was of the size of a small nut, prolonged in the course of the vessel, and very hard. The cavity was filled with fibrinous clots adhering to the parietes. The left vertebral artery was nearly obliterated, and the left communicating artery was dilated, while the right was narrow. The vessels were atrophic	—	Adhesions between the surfaces of the arachnoid, and great vascularity of the pia mater. Numerous dilatations of the vessels of the pons	—	F., ast. 61, in service of M. Frémyn at the Hôtel Dieu, long suffered from impairment of intelligence and motor power, with involuntary passage of evacuations, and had an old goitre. Two months before death had fallen on left side and lost the use of the left arm. When admitted had partial hemiplegia of left side. She died comatose in four days.
Brit. Med. Journ., vol. ii, 1870, p. 87, Dr. Russell, Birn. Gen. Hospital	The aneurism was an inch and a half long and an inch wide, and there was also a smaller sac on a branch of the right case at middle cerebral. The basilar sac contained firm laminated conglom.	—	Superficial softening of the pons and medulla	—	F., ast. 26, had chance six years before followed by sore throat. Had two distinct attacks of hemiplegia, and finally became paraplegic.
Amer. Jour. of Med. Sci., N.S., vol. The aneurism arose from the right side of the vessel, and was rent in the sac extending along the	There was a large extravasation of blood	There was a considerable rent in the sac	—	—	M., ast. 87, subject to severe paroxysms of pain over the right orbit and at the back of the neck. This was succeeded by pain in the

XIV, 1872, about half an inch in  
P. 375, Dr. Bartholow, its longest diameter.  
The coats of the artery of Ohio; were atheromatous and  
occurred in right vertebral was very  
the practice small of Dr.  
Johann, of Cincinnati

flature of Sylvius, and  
covering the pons and  
medulla, and filling the  
fourth ventricle. There  
was fluid in the ventri-  
cles, and the sixth  
nerve was pressed upon,  
and the fifth also in-  
volved in the disease

eyeball, numbness of the right side of the  
nose and lip, and proosis of the right eye.  
He partially recovered the power over the lid,  
but there was also convergent squint of the  
right eye and severe pain at intervals. For  
about two years before death he had numbness  
of the left arm and hand, and his character  
became fickle and strange. He had had sun-  
stroke twelve years before.

### Aneurism of Cerebellar Arteries.

Path. Tr., The sac occupied the  
vol. x, right superior cerebellar  
1858-59, artery, and was situated  
in the substance of the accidentally in  
hemisphere. It was about the size of brain  
nearly twice the size of brain  
a grain of wheat. It  
was fusiform in shape,  
and the coats were  
thickened and hardened  
and contained athero-  
matous and earthy de-  
posits. It gave off  
several small branches  
perfectly ossified. The  
anterior extremity was  
continuous with the  
right superior cere-  
bellar artery, of which  
the coats were thin, but  
otherwise natural. The  
other arteries of the  
brain were athero-  
matous

The sac was  
entire, and serum on the surface of  
the brain and in the ventricles. There was  
the small spongelike cyst  
in the right corpus  
striatum

M., set. 56, was under treatment in St.  
Thomas's for gout or rheumatism. Five days  
after admission he was seized with severe  
epigastric pain and vomiting, and became  
comatose, and died in two days.

The heart was  
hypertrophied,  
and weighed 17  
oz. There was  
no valvular dis-  
ease. The kid-  
neys were small,  
and the surface  
smooth and mol-  
tled, and con-  
tained few cysts  
and deposits of  
gritty yellowish  
particles. There  
were gouty de-  
posits in the  
joints. The  
coats of the  
aorta had nu-  
merous bony  
plates and  
patches of athe-  
romata

Reference to author.	Seat, form, and size.	Result.	State of brain, &c.	State of the other organs.	History.
Brit. and For. Med. Chir. Rev., vol. xxxvi, July to Oct. 1861, p. 491.	Left, anterior cerebellar artery; aneurism solid, filled with dark coagula and decolorised fibrin. Capillaries of brain in state of fatty degeneration	—	Arachnoid thickened at places and effusion under it and in ventricles; vessels congested. Tumour pressed on left middle crus cerebelli and pons and left lobe of cerebellum, and the substance of brain in these situations superficially softened. The root of fifth nerve pressed on, and seventh of left side stretched. Optic nerve, commissure, and tractus dwindled and softened, of a somewhat yellow colour and semitransparent	—	F., <u>at. 46</u> , about five years before death had pain at the front and top of head, with dimness of sight and occasional momentary blindness, and sense of heaviness and stupor. Two years after began to have epileptic fits, and the following year became quite blind, and there was paralysis of the left side of the face, deafness of the left ear, and loss of smell in left nostril, the paralysed parts being exquisitely sensitive. The pupils were dilated and fixed, but there was not ptosis. Memory occasionally defective, but mental power not usually impaired. To these symptoms succeeded want of power over muscles of left side of neck, and afterwards undue sensitiveness of paralysed parts, and twitching of left arms and right side of neck and shoulders, and stiffness of left side. She lost the sense of taste on left side, and became drowsy, and she died two years afterwards in a fit.
Bullet. de la Soc. Anat. de Paris, xxix, Année 1864, 2me série, p. 485, and atheromatous. The M. Fernet aneurism was situated at the side of the bulb	The aneurism was situated on the inferior artery, and was the size of a pea. The other arteries of the brain were altered	The sac was ruptured	There was hemorrhage which had separated to a great extent the visceral arachnoid	—	F., <u>at. 60</u> , brought into Jervis Street Hospital in state of insensibility. Was in usual health, when, in preparing breakfast in the morning suddenly seized with fit and fell down insensible. When admitted she was
Dub. Jour. of Med. Sci., 1875, p. 373.	Aneurism of superior cerebellar artery. It was of large size, and Jan. & June, there were smaller dilatations of vessels of pons, with breaking	Blood-vessels injected, and recent lymph on arachnoid.	—	Heart extensively diseased; right auriculo-ventricular contraction of	

Dr. Mac- Swiney, in Rep. of Dub. Path. Soc.	up of pons and of some of the branches of the basilar artery	tissue of brain. cular aperture and of aorta, partly open, pupils very much contracted, and with vegetal-limbs generally relaxed; died in eight hours.
		Aorta dilated and surface atheromata

### Aneurism of Posterior Cerebral Artery.

Bullet. de la Soc. Anat., xvi, année 1842, p. 112, M. Delpech	The aneurism formed a tumour the size of a pigeon's egg, between the chiasma of the optic nerve, the middle lobes of the brain, and the peduncles and the protuberance. It was ovoid, firm, and contained dark coagulum and fibrinous concretions	The third nerve on the left side where it passes from the cerebrum was entirely destroyed, and beyond that point was reddened and softened. The fifth pair was less compressed, but was reddened and softened where it crosses the peduncles before reaching the ganglion of Gasser. There was a deep depression in the protuberance	—	Heart healthy	F., sit. 20. Had had syphilis. Seized while at stool, with insensibility, and when admitted into La Pitié ten months after, there was ptosis of the left eyelid, and the ball of the eye was forcibly fixed in the external angle. The pupil was greatly dilated and fixed. There was also severe pain in the temporal and supra-orbital region, and afterwards loss of sensation in left cheek. These symptoms were followed by epileptic convulsions, which though she was relieved by bichloride of mercury, ultimately proved fatal after about four months had elapsed. The right limbs were contracted and painful.	M., sit. 56. Had suffered at intervals for twelve years with gout, and three months before his death was under treatment at St. Mary's for that disease. About a week after his admission he had headache and giddiness, especially on rising from the horizontal position, and it appeared that he had had similar symptoms occasionally for a year. He was relieved, and went on only suffering from weakness. About six weeks after, he was suddenly seized with symptoms of apoplexy when in the street, and was taken to the Middlesex Hospital, but died in the waiting-room.
Path. Tr., vol. vii, 1855-56, p. 129, Dr. Van der Byl	The aneurism arose from the artery of the right side, where it enters the middle cornu of the ventricle. It was the size of a hen's egg, and contained concentric fibrinous layers, some dark, some light coloured	A slight rupture and limited extravasation had occurred before the last attack and more extensive	A slight rupture and much congestion, and the convolutions were flattened, but the substance of the brain was pale. There was bloody serum and clots in both ventricles. The aneurism projected into the middle or descending cornu of the right ventricle, and the brain substance around was broken up and presented a yellowish granular appearance, mottled with pink spots	—	—	—

Reference to author.	Seat, form, and size.	Result.	State of brain, &c.	State of the other organs.	History.
Path. Tr., vol. viii, 1856-57, p. 166, Mr. Squire	The aneurism arose from the artery on the left side, near the basilar artery. It was about the size of a large walnut, and was dense and firm, and of a red colour, and was filled with dense, yellowish fibrin. The basilar was very much reduced in size, and there were several anastomosing vessels. The coats of the vessels were thin, but not otherwise changed. The aneurism was 4 inches in its transverse circumference and 3½ in its antero-posterior. There was a cavity containing soft coagula.	—	The tumour pressed up the floor of the third ventricle, rising between the thalamæ, and occupied the space in front of the pons and between the crura cerebri. The third nerve adhered to the base of the tumour	—	M., est. 40, deserted by her husband, and left to support three children. When seen she laboured under adynamic fever, probably from destitution. She was not comatose or paralysed, and the sense of sight and hearing, and the power of deglutition, were not interfered with.
Pennsyl. vania Hosp. Reports, vol. ii, 1859, p. 83, Dr. J. H. Hutchinson	The aneurism was situated on a branch of the artery of the left side, and was about the size of a fibert. The other vessels of the brain were healthy	The sac had ruptured	Vessels engorged and coagulated blood in the lungs ventricles and at the aorta base, and fluid in the ventricles. The brain substance around the aneurism at the posterior part of the left lateral ventricle was broken down	Heart and lungs healthy and at the aorta not dilated. Liver and spleen somewhat enlarged.	M., est. 14, a patient in the Episcopal Hospital in 1868. Four days before admission he had two slight convulsive attacks followed by pain in the limbs, and he was said to have had oedema at the lower extremities. About six weeks before these attacks he was noticed to have become languid and indisposed to exertion. He had also almost constant headache and frequent epistaxis. Soon after his admission he had a return of the convulsive attacks with symptoms of fever and

rise of temperature, and his head was retracted, and there was pain in attempting to move it. The pupils were dilated and partially insensible, and he had pains in the back and limbs. He died comatose in about ten days.

M., s<sup>t</sup>. 35, patient in Richmond Hospital in 1836, found two days before lying insensible in the street, and when admitted was semi-comatose, but capable of being aroused for a moment; stertor, pupils dilated. Left arm and both lower extremities paralysed, involuntary discharge of urine and feces; remained much in the same state, and died in five days.

**Prov'd fatal**  
Aneurism of apparently the posterior cerebral artery, forming an oval solid tumour 1 $\frac{1}{4}$  and 1 $\frac{1}{4}$  in. in diameter. It contained concentric laminae of coagula to a certain extent deprived of colour, and the opening into the sac, the artery was also occluded.

Bullet. de la Soc. Anat. de Paris, xlii, p. 257, M. Victor Hanot

The aneurism was on the left posterior cerebral artery, and was 6 cm. long and 3 broad, and of irregular shape. It extended from the anterior part of the protuberance to the chiasma of the optic nerves, and from one sphenoidal lobe to the other. It contained whitish stratified clot with some recent coagulum. There were slight atheromatous patches in the basilar and other vessels.

The sac had ruptured profusely into the middle ventricle, and pushed aside the right optic nerve, and the chiasma, and left optic nerve were compressed. The motor oculi and the bulbar nerves were not interfered with, nor was the spinal cord

**F., s<sup>t</sup>. 50**, in the service of M. Voisan. She had suddenly become deaf ten years before. Two years after that she had diplopia and amblyopia of the left eye, and in a month lost the sight of the eye entirely. She then also began to have epileptic attacks every month or two, but without general convulsions. In the intervals she appeared well except having some slight headache and singing in the ears. Eighteen months before her death she became blind of the right eye, and her intelligence was much impaired. Though deaf and blind the senses of taste and smell were unaffected, and the limb were not paralysed, and the sensation remained. Latterly she was quite fatuous.

The heart was healthy, but there were alternating patches of the thoraic aorta. The lungs, spleen, and kidneys were healthy

## MULTIPLE ANEURISMS.

## Vertebral, Inferior and Anterior Cerebellar, Right Sylvian, and Posterior Communicating Arteries of both sides.

Reference to author.	Seat, form, and size.	Result.	State of brain, &c.	State of the other organs.	History.
Ibid., obs. vii., p. 38. M. Bourneville. Bull. Soc. Anat. de Paris, t. xiii., anno 1868, p. 449	Aneurism of the right vertebral artery, arising about 2 cm. below the origin of the basilar. It was ovoid, communicating with the anterior cerebellar artery about 1 cm. long and 6 mm. wide; another on the inferior and anterior cerebellar artery of the same side, fusiform, 7 mm. long and 6 mm. wide. Basilar and vertebral arteries and branches only slightly atherosomatous, and the vessels nearly of the proper size. The right Sylvian artery was dilated and the vessels which arose from the carotid were slightly atherosomatous. There were also aneurisms of both posterior communicating arteries. That of right 6 mm. long and 4 wide; that of left 1 em. long and 8 mm. wide. The sac was thickened, and contained stratified fibrinous clots	The aneurism of the blood at base around the left posterior olfactory nerves and left cerebral peduncle and anterior pyramid, patches and in cerebellar pia, plates, especially where the vessels joined the ventricles. Small aneurisms in convolutions of the brain, and in the corpus striatum, and extravasations therein	Left ventricle hypertrophied with incomplete hemiplegia of right side without contraction. Fracture of femur. In 1867, after having had vague pains in the head, was taken with an apoplectic attack. She partially recovered and then had another attack, and after about eighteen months a third, which proved fatal.	F., est. 53. In Salpêtrière under M. Charcot, with incomplete hemiplegia of right side without contraction. Fracture of femur. In 1867, after having had vague pains in the head, was taken with an apoplectic attack. She partially recovered and then had another attack, and after about eighteen months a third, which proved fatal.	

For the conclusion of Dr. Peacock's paper see p. 317.

## ERRATA.

For Mr. Stevens, p. 14, line 15, read Mr. Stewart.  
 For de\*, p. 22, line 24, read du.



## EXPLANATION OF PLATES I AND II.

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### PLATE I.

Fig.

- 1.—Aneurism of the left middle cerebral artery (Case 1, p. 181).
- 2.—Aneurism of the right internal carotid immediately below the origin of the middle cerebral (Case 2, p. 184).

### PLATE II.

Fig.

- 3.—Middle fossa of the skull. On the posterior surface of the right petrous bone the elongated openings into the tympanum are represented (Case 1, p. 182).
- 4.—The appearance of the under surface of the brain in Case 1, described in pp. 181 and 182.

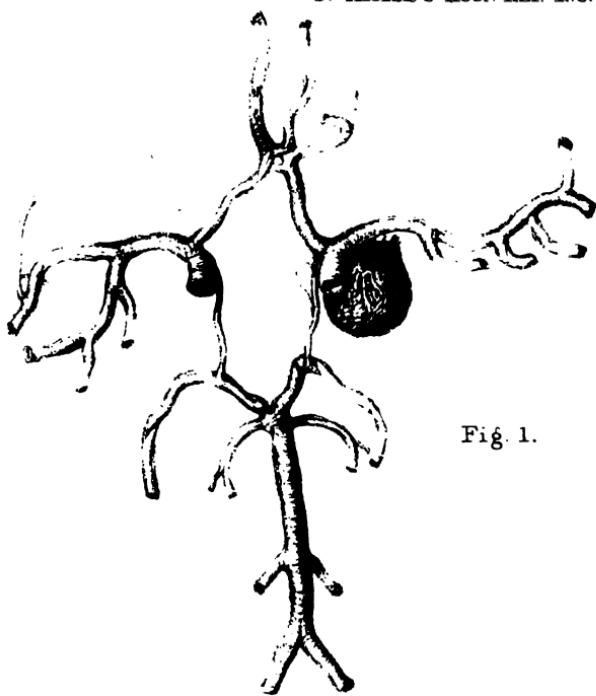


Fig. 1.

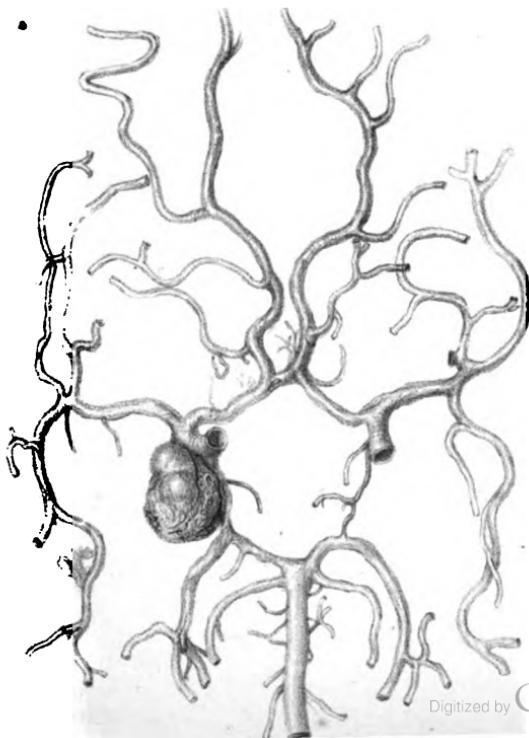


Fig. 2.



Fig. 3.

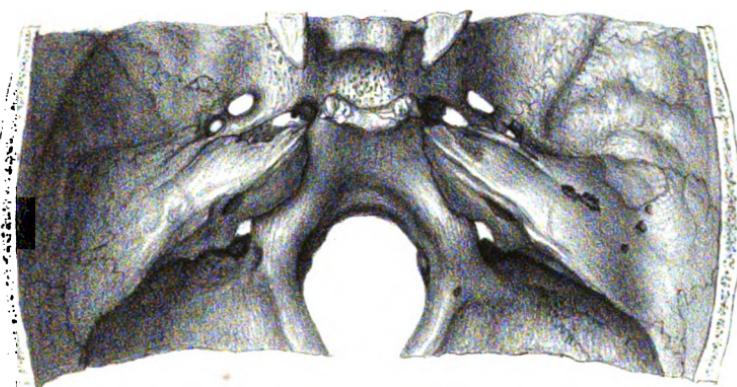
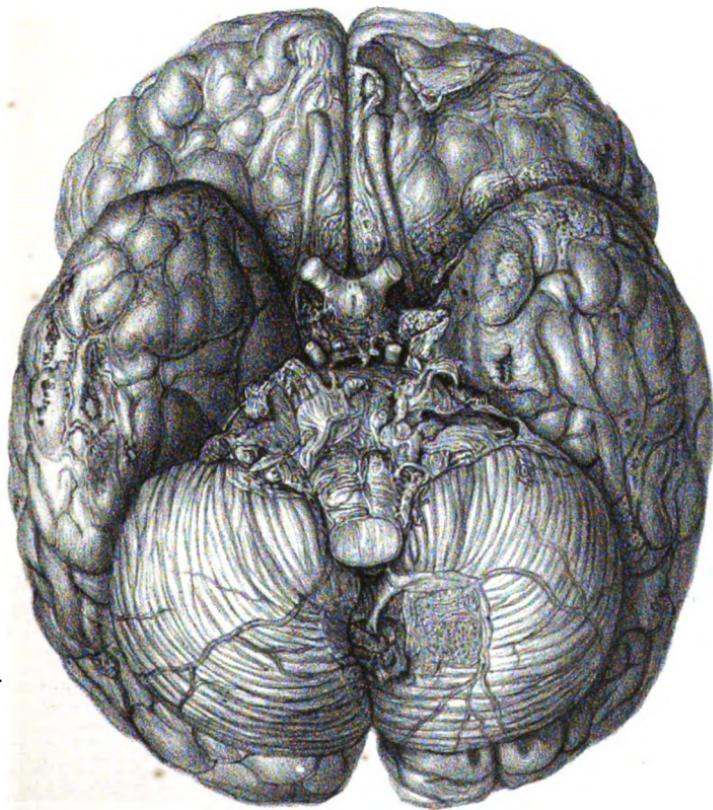


Fig. 4.





# ON THE "PRESYSTOLIC" BRUIT.

WITH CASES.

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THAT the "presystolic" bruit, in the great majority of cases in which it is observed, is associated with a constriction of the mitral orifice, will be admitted by every one; but that there are important exceptions to this rule is a fact which I believe to be equally certain, as attested by the observation of two exceptional cases, reported by Dr. Austin Flint in the 44th volume of the 'American Journal of Medical Science,' and of some cases of a similar nature which have occurred during the past year (1876) in the wards of St. Thomas's Hospital, and which form the principal basis of the present communication. Whilst, however, there is agreement as to the diagnostic value of this bruit as a general rule, the exact nature and cause of the bruit itself are still matters of uncertainty. The hope of being able to throw some light upon this subject, and the important bearing which the cases referred to seem to have upon it, have led to the present attempt at its elucidation.

Regarding the bruit, in accordance with its accepted pathological significance, as a fluid murmur produced at the mitral orifice, the question to be considered is reduced to a

discussion of the views advanced in explanation of it, on the one hand by M. Fauvel and Professor Gairdner, and by Dr. Barclay on the other. There is no room for any other hypothesis than that a fluid bruit, at this particular period of the heart's action, is produced at the mitral or tricuspid orifice, either by the direct auriculo-ventricular blood current at its conclusion, or by the occurrence of regurgitation at the very commencement of the ventricular systole.

The facts discussed in the following pages in their bearing upon this question have been included under four heads:

I. Certain clinical observations in connection with the occurrence of the "preystolic" bruit in the presence of contraction of the mitral orifice, considered in conjunction with physiological facts to which they are related.

II. Cases in which a "presystolic" bruit has been heard, but in which the autopsy has shown that no appreciable narrowing of the mitral orifices had existed.

III. Cases in which a bruit similarly related to the second sound, and of a character identical with that of the "preystolic" bruit, has been heard at the base of the heart.

IV. Certain facts connected with the "presystolic" bruit, which have been regarded as more especially opposed to the views advanced by Dr. Barclay.

And here in the first instance I must observe, that what I cannot but consider the somewhat too ready and general acceptance of Professor Gairdner's views, has led to the use of the term "presystolic" in a rather loose and inaccurate manner, a result which appears to me to have tended to keep out of sight the chief difficulties which his hypothesis presents. The term "presystolic" bruit, in its limited and conventional sense, as an abbreviation of "*pre-systolic sound*" bruit, is a perfectly accurate designation of the bruit in question, and in this precise and limited sense I shall uniformly employ it throughout the present paper. But when the designations of "auricular systolic," or "direct mitral" bruit, (as suggested by Professor Gairdner,) are received as synonymous with it, this conventional limitation of its meaning becomes unnecessary, and the term "presystolic" is taken in its literal sense. But until it is shown conclusively that, under the conditions presented, there is no appreciable interval between the auricular

systole and the first sound of the heart, this use of the term is scarcely warranted. Until this is proved, there still remains not only the fact that, normally, such an interval does exist however short, but also the possibility that this brief interval may be increased under the abnormal condition found.

Without previously clearing up this point, there are no sufficient grounds for saying that we have only to determine that a murmur is presystolic, and that it does not accompany the second sound, to recognise it as a mitral direct murmur; or that, when the first sound is heard in the middle of an apparently continuous bruit, we shall "then know absolutely that the first part, preceding and running up to the first sound, must be auricular-systolic, and that the part succeeding the first sound must be ventricular-systolic,"<sup>1</sup> or that a similar inference is to be drawn from a change of character in the middle of such a bruit, from a harsh to a blowing quality, even when no first sound can be distinguished. Supposing that, with the mitral curtains rigid and unyielding, but still capable of being brought into partial contact by a pressure below that which the ventricle is exerting, the ventricle should at the same time be acting in an abnormally deliberate and sluggish manner, we should have conditions in which regurgitation occurring at the commencement of the ventricular systole, would produce a harsh and rasping bruit of the most intense degree, but in which, after the curtains have come into contact, and after their vibrations have thus become deadened, and the regurgitant stream very greatly reduced, the final part of the bruit would be of a modified and more blowing character.

Open to the same objection is the line of argument of another authority on the subject. Dr. Mahomed, having adduced evidence of an increased period of the auricular systole in cases of mitral obstruction, by means of cardiographic tracings, (and I shall have occasion to refer to these more particularly further on), observes that, if evidence of the prolongation of the auricular systole is found, Professor Gairdner's argument must be admitted,<sup>2</sup> and he contends that these results of his cardiographic observations completely overthrow Dr. Barclay's position. But, in considering this

<sup>1</sup> Gairdner, 'Clin. Med.', p. 577.

<sup>2</sup> 'Med. Times and Gaz.', 1872, vol. i, p. 715.

question, it is certain that the *position* of the auricular systole in the cardiac rhythm is of far more importance than the amount of *force* which it is capable of putting forth, the peculiar and intimate relation of this bruit to the first sound being a much more characteristic feature of it than the loudness or duration of the sound produced. So far from agreeing with Dr. Mahomed's inferences from the character of the cardiographic tracings in question, I regard them, as I shall endeavour to point out a little further on, as affording evidence which is quite opposed to Professor Gairdner's hypothesis, but which is, on the other hand, fully in accordance with the view adopted by Dr. Barclay.

As a matter of fact, the substitution of the literal for the conventional meaning of the term, has led to the designation as "presystolic" of any bruit terminating with the first sound, or indeed to a purely diastolic mitral murmur separated by a distinct interval from it. And this is natural, since in any case an obviously diastolic mitral bruit must necessarily be "direct" in its nature, and will, on this hypothesis, be identified with the typical short "presystolic" bruit, by which it is commonly followed. The continuity of the murmur when a prolonged bruit is heard, covering the entire diastolic period, and finally terminating abruptly in the first sound, may indeed convey the impression that the bruit is one and the same, and that the whole must be produced by one continuous blood stream, but it does not necessarily follow that this should be the case. If it were so, this would be at once conclusively fatal to Dr. Barclay's hypothesis, since no one can suppose that mitral regurgitation could produce a bruit commencing immediately after the second sound. There is no difficulty, however, in conceiving that, under certain conditions, a long and harsh diastolic bruit, occupying the entire diastolic period, and followed by a bruit of regurgitation commencing immediately with the ventricular contraction, might be indistinguishably merged in the latter. Of the manner of production of the first part of such an apparently continuous bruit there can be no question, and its character seems to leave little doubt of the sufficiency of the pressure of the blood accumulated in the auricles to produce a bruit independently of the force of the auricular systole,

the effect of which may often be distinguished as an intensification of the murmur towards its conclusion, just before the "presystolic" termination. It is with this last, the typical short, harsh, and abruptly terminated "presystolic" bruit, that I am now alone concerned.

*I. Facts observed in connection with the occurrence of the "presystolic" bruit in the presence of contraction of the mitral orifice.*

In proceeding to consider the evidence of physiological and clinical facts as to the production of such a bruit, in the presence of mitral obstruction, in the manner contemplated by Professor Gairdner and Dr. Barclay respectively, two questions suggest themselves :

*a.* What modifications in the normal character and mutual relations of the cardiac phenomena are necessarily implied in each hypothesis ; and how far can the occurrence of mitral stenosis be regarded as tending, directly or indirectly, to produce such modifications in the cardiac function ?

*b.* What evidence do clinical observations afford of the actual presence of such modifications of the heart's action, in cases where the existence of this condition of the mitral orifice is indicated ?

1. *a.* Taking first the "auricular-systolic" hypothesis of Professor Gairdner, it will be seen at once, from a consideration of what is known of the relation of the normal auricular to the ventricular systole, that the former, however powerful, could not produce a bruit of such a character, under any condition of the mitral orifice. Direct observation of the heart exposed in living animals has shown, that the auricular systole, though rapidly followed by the ventricular contraction, is quite distinct from it, and Dr. Hayden in the account of his examination of M. Groux, describes the pulsations of the upper and lower tumours, which (in this peculiar case) corresponded to the right auricle and ventricle respectively, as being quite distinct in their succession.<sup>1</sup> But how very considerable an interval normally exists between the moment

<sup>1</sup> Hayden, 'Diseases of Heart and Aorta,' p. 92.

of *maximum* intra-auricular pressure, which could not occur before the climax of the auriculo-ventricular influx, and the cardiac impulse, is to be inferred from the results of M. Marey's experiments. The simultaneous tracings taken by him show that the interval between the summit of the auricular curve and the first summit of the apex tracing is equal to one sixth of the period of an entire revolution of cardiac movements, and equal to half the whole period of the ventricular systole, or, what comes to nearly the same thing, half the interval between the first and second sounds.

Hence the auricle, contracting in its normal rhythm, would produce a "bruit de soufflet" of the character of the mitral systolic murmur, the climax of which would be separated from the impulse or first sound by such an interval. It could not produce a bruit approaching in character to that presented in the "presystolic" bruit.

Nor could this character be assumed, in any degree, through the permanence of the auditory sensation produced by an auricular systolic bruit, over the interval existing between the climax of the auricular contraction and the first sound; for a bruit which, like the "presystolic" bruit, becomes gradually intensified up to its very termination, is referable only to a cause simultaneously and correspondingly increasing in power. A merely persisting impression would be a more or less rapidly fading impression, and could not have an apparent increase of intensity. Hence, in order that a bruit, becoming louder up to the very moment the first sound is heard, should be produced by the auricular systole, this must be only just reaching its climax at that instant; and further, in order that the bruit should be abruptly terminated at this moment, the intra-ventricular pressure must rise with such rapidity, as to arrest the auriculo-ventricular influx at the very height of its force, and close the mitral valve, in an inappreciable period of time, too short to allow any tailing off of the bruit to be heard. Professor Gairdner's hypothesis implies the assumption, that the occurrence of mitral stenosis alters the relation normally obtaining between the auricular and the ventricular systole in the manner indicated, and produces conditions under which the effect of the latter is brought into action in this sudden and impulsive manner.

As far as concerns the mere possibilities involved in the physical conditions of the cardiac action, there would perhaps appear to be no great difficulty in conceiving how such a result might be brought about. The occurrence of a narrowing of the mitral orifice tends directly to induce dilatation and hypertrophy of the left auricle, and a prolongation of the period of its contraction; it is possible that this might be so prolonged as to be only just reaching its climax at the moment when the auriculo-ventricular influx is checked by the force of the ventricular contraction. And further, the ventricle being imperfectly filled with blood owing to the obstruction behind, it might be supposed that the intraventricular pressure would not be affected appreciably at the commencement of its contraction, and that this would therefore, at first, have no influence in checking the influx through the mitral orifice. Supposing this to be the case, and that the ventricle did not *grasp* its contents until a later, and more rapid, period of its contractile movement, it would then exert a proportionately sudden and impulsive compression upon the blood within it, and, by causing a sudden rise in the intraventricular pressure, it might thus so abruptly arrest the inflowing stream from the auricle, and so immediately close the valves, even if so rigid as those commonly seen in cases of mitral obstruction, as to produce an effect corresponding with the peculiar abrupt character of the typical "presystolic" bruit.

b. Admitting, then, that the occurrence of mitral obstruction would be capable of giving rise to a "presystolic" bruit in the manner contemplated by Professor Gairdner and M. Fauvel, the further question has to be considered, as to whether there are any clinical or other facts supporting, or otherwise, the assumptions which are implied in it, that is to say, the postponement of the climax of the auricular systole to the very latest period, and the sudden and abrupt arrest of its effect by the ventricular systole.

The evidence of facts bearing upon the second of these points being, as it seems to me, of a less conclusive character than that adducible in the reference to the former, I will consider that first.

To account for the sudden arrest of the auriculo-ventricular blood stream, in a manner corresponding with the rhythm

and character of the "presystolic" bruit, there are, as just indicated, two possible factors available, 1, a state of imperfect repletion of the ventricle, and 2, an increased rapidity of its contraction. Of the former of these nothing very definite can perhaps be said, as there is nothing to indicate the degree of "active dilatation" which the ventricle experiences from the permanent elasticity of its walls, and the effect of distension of the coronary arteries. Supposing these forces to be great, and the impediment to the influx of blood to be great also, it is possible that the wall of the ventricle at the commencement of the systole might be flattened, or even depressed, and that considerable contraction might occur before it assumed a rounded form, and before it could grasp and compress its contents.

But, in the presence of a gradually occurring impediment, the size of the ventricle would become adapted, more or less accurately, to the diminished volume of blood it has to act upon at each contraction, and it is scarcely conceivable that any great disproportion should exist in this respect, except perhaps when its action is greatly accelerated by excitement.

Of the second factor tending to the same result, an increased vigour and rapidity in the ventricular systole itself, more may be said. From the observation, that when the ventricle has become much hypertrophied from impeded action, and from the resulting tendency to overdistension, it contracts in a slower and more deliberate manner, the inference has been drawn, that under the opposite conditions, presented in cases of mitral constriction, an opposite effect would be produced; and Dr. Galabin has called attention to the vertical character of the systolic upstroke, seen in most of the cardiographic tracings taken by him from cases of this kind, as confirming this supposition.

But it must be observed, that the sharpness of this upstroke indicates only the abruptness, with which the contracting ventricle presses against the wall of the chest, *i. e.* the suddenness with which its walls become tensely stretched upon the blood within it. If the ventricle is contracting in a sluggish manner, and if the mitral curtains are rigid, or so unfavorably situated as not to respond to the pressure at first brought to bear upon them, but permit the blood to flow back into the

auricle, there will be no great tension of the ventricular walls so long as this free reflux continues, but if, under the gradually increasing intra-ventricular pressure, the resistance of the unyielding curtains is eventually overcome, and the valve closes quickly and completely, (and this will occur more abruptly in proportion to the degree of force required to be exerted), the intra-ventricular pressure and the tension of the ventricular wall will, at this moment, be increased with corresponding rapidity, and thus might be produced a systolic upstroke in the cardiographic tracing as sharp as that seen in the tracings referred to.

This character of the apex traces cannot then, as it seems to me, be regarded as affording any great support to the inference mentioned. But that inference, if unsupported by other facts, can scarcely stand alone. It does not seem to follow from the observed tendency of ventricular embarrassment, that this organ when imperfectly supplied will tend to act with a corresponding increase of vigour, for it might be anticipated with equal likelihood, that a diminution of the stimulus of due repletion, and a diminution at the same time of the work required of it, would tend to cause a corresponding *diminution* of its activity. In favour of this view of the case is the fact, that in cases of mitral obstruction the heart's action is often remarkably deliberate and slow, while on the other hand, overdistension of the ventricle is wont to give rise to an exceeding perturbation and rapidity of its action, and a similar jerky character of the contraction of the overloaded right ventricle is evidenced in the character of the systolic pulsation, sometimes visible in the veins of the neck.

There does not, then, appear to me to be much clinical evidence of the existence, in cases of mitral stenosis, of a degree of vigour in the ventricular contractions such as would tend to cause a very sudden arrest of the auricular-systolic influx of blood into the left ventricle.

The evidence of clinical facts in opposition to the other requirement of Dr. Gairdner's hypothesis, that is, the occurrence of the auricular systole at the very end of the long pause, in the strictly "*presystolic*" period, seems to me to be of a much more definite and conclusive nature.

It may be pointed out in the first place, that such a post-

ponement of the auricular contraction would not merely be attended by some waste of the auricular force, increased, in accordance with an especial provision of the economy, to counteract in some degree the effects of this impediment to its action ; but that it further implies a misdirection of much of this augmented force in a manner which is directly detrimental. If the auricular systole occurs at the very latest period of the long pause, and in such a manner that its greatest force is only just being exerted, at the instant that its effect is suddenly and completely destroyed by the opposing force of the ventricular contraction, the remainder of the force put forth by it will not only be entirely inefficacious in aiding the forward movement of the blood, but will be exerted in increasing the pulmonary congestion, which is a serious complication in the disease in question, and one which, by efficient action, this power is calculated in great measure to relieve. A waste or misdirection of the increased auricular force seems the less likely to occur under conditions in which the heart is observed to act with much deliberation and regularity. To suppose that the auricular systole may be thus postponed is, however, opposed to several facts which indicate that, so far from this being so, the auricular systole is, in these very cases, separated by an abnormally distinct interval from the succeeding first sounds.

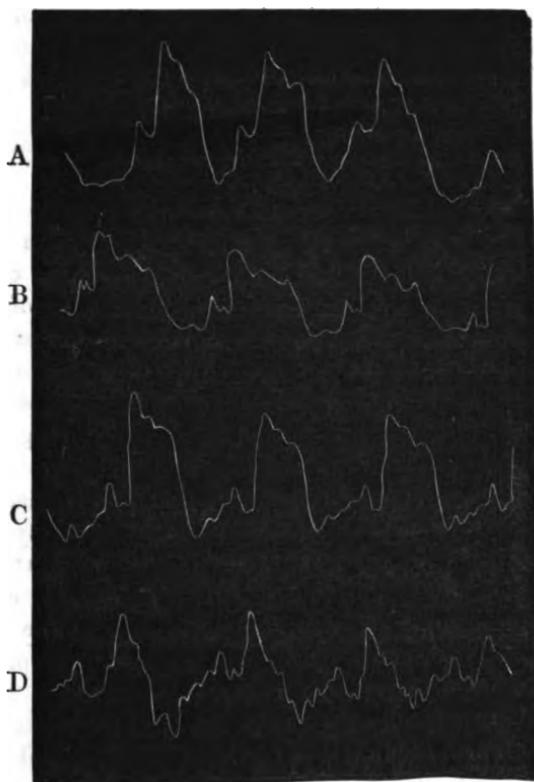
If we might draw any conclusions as to this from the action of the right auricle under analogous conditions of embarrassment and overdistension, the distinct interval between the systolic and diastolic pulsations seen in the veins of the neck in cases of pulmonary embarrassment, would indicate that there is a very sufficiently marked separation between the auricular and ventricular contractions under such conditions.

In regard to facts more directly affecting the question of the rhythm of the contraction of the left auricle in cases of mitral obstruction, it may be mentioned that Dr. Fagge, in the collection of cases of "presystolic" bruits published by him in the 16th volume of the third series of the 'Guy's Hospital Reports,' records two cases in which a peculiar wavy diastolic bruit was heard at the apex, which he regarded as probably indicating an occurrence of the auricular systole at the very commencement of the diastolic interval, as a result of

great embarrassment of the heart's action through mitral stenosis.

The most distinct evidence on this point, however, may, I think, be deduced from the characters of the cardiographic tracings taken by Dr. Galabin and Dr. Mahomed from more ordinary cases of the form of disease, in which bruits of less complicated character were heard.

The following are copies of cardiographic tracings taken by Dr. Galabin, and published by him in his paper "on the interpretation of cardiographic tracings," contained in the 20th volume of the same series of 'Reports.' (Plate III., figs. 9, 10, 15, 19.)



The first two tracings were taken from patients in whom a properly "presystolic" bruit was heard; in the first case this

was accompanied by, and apparently continuous with, a diastolic apex bruit commencing from the second sound; in the second case a "presystolic" bruit alone was heard. The two latter tracings were taken from cases in which a diastolic bruit was heard commencing from the second sound, but ceasing before the first sound.

By a comparison of the above figures it is seen, that the highest summit of the diastolic portion of the tracing is, in each case, separated by a very conspicuous interval from the main upstroke, which indicates the moment of the impulse and first sound (Fig. c).

This feature is pointed out, both by Dr. Galabin and by Dr. Mahomed, as especially characteristic of cardiograms taken from cases of presystolic bruit, being indicative of an earlier and more powerful contraction of the auricle than normal, and it is a feature which these cardiographic tracings from such cases generally present in greater or less degree. The above tracings are some of those indicated by Dr. Galabin as presenting most distinctly this especial character.

If the typical "presystolic" bruit, running up to the first sound, which was heard in the patient from whom the tracing **B** was taken, was really produced by the auricular contraction, the elevation in the auricular-systolic portion of the trace could not have here indicated the rise and fall of the intra-auricular pressure during the contraction of that cavity, as it is seen that it should do, under normal conditions, by a comparison of Marey's simultaneous tracings of the apex beat and of the intra-auricular pressure. The inference implied by this hypothesis would be, that the first and highest elevation of the auricular portion of the trace can merely have indicated the suddenness of the commencement of the auricular systole, and that the subsequent part of the contraction, though more powerful in correspondence with the intensification of the bruit, must have failed to be duly represented on account of its more gradual occurrence. Such a supposition, however, is negatived by a consideration of the tracings **c** and **d**, which are quite similar to **B** in this feature, in relation to the especial clinical phenomena presented by the patients from whom they were taken.

In the cases from which the tracings **c** and **d** were taken,

no bruit was audible in the proper "presystolic" period, though in each case a prolonged and harsh diastolic apex bruit was heard, indicating with certainty a great degree of mitral stenosis. In such a condition of the mitral orifice it is certain, that an auricular contraction capable of producing an elevation in the apex tracing, such as is seen in the auricular systolic portion of these tracings, must have produced a very distinct increase in the loudness of the continuous direct mitral bruit, which was heard extending over the greater part of the diastolic interval. Hence this bruit must have had an intensification of its loudness, at a period corresponding to the auricular systolic elevations of the tracings, and of a degree proportionate to their prominence; an intensification of the sound which would have been of a temporary character if, in these instances of mitral stenosis, the length as well as the time of the auricular contraction were truly represented in the cardiograph, but which, if continued up to the apex beat, would have confirmed the assumption implied in the auricular-systolic hypothesis. The fact that in both instances the bruit ceased before the first sound, and that in both cases the occurrence of just such a temporary, penultimate, intensification of the bruit was especially noted, seems to prove that here, in the presence of mitral stenosis of a high degree, as indicated by the harshness of diastolic bruit throughout, the auricular systole is faithfully represented in the cardiogram, as is demonstrated to be the case under normal conditions.<sup>1</sup>

<sup>1</sup> I believe from my own observation, that a distinct intensification of the prolonged compound diastolic and "presystolic" bruit, just at the conclusion of the diastolic portion of it, is not unfrequently present, a character which I represent in Fig. 1, given below. I was not a little interested to find that in the diagram of this bruit in Professor Gairdner's work, reproduced in Fig. 2, a similar intensification is indicated as occurring about the middle of it, though I do not find any special reference to the point in the text.

FIG. 1.



The shading represents the sounds as heard; the dotted lines indi-

One other possibility alone seems capable of invalidating the inference drawn from the above facts, the possibility that the first and principal elevation of the auricular-systolic trace may have been produced by the contraction of the right auricle instead of by that of the left, as has been tacitly assumed, the former having been left out of account. It might be thought that the narrowness of the mitral orifice, on the one hand, causing more or less embarrassment of the right heart, and tending, on the other hand, to interfere with the transmission of the effect of the contraction of the left auricle to the apex of the ventricle, would give an *a priori* probability to such a supposition. But, in regard to the second point, it may be pointed out, that a state of imperfect repletion of the left ventricle must present a condition favorable to the projection of the narrow and forcible blood stream, issuing from the contracted mitral orifice, directly against the apex of the ventricle, in a manner that might well account for the distinctness of the elevation by which its effect appears to be represented. The difficulty here presented seems, however, to be removed by a comparison of cardiographic tracings taken from cases attended by as considerable, or even greater, distension of the right auricle, as from cases of mitral insufficiency, with those above given. In examining the series of such cardiographic tracings comprised in the plates appended to Dr. Galabin's memoir already referred to, it will be found that this very remarkable interval between the first auricular-systolic elevation and the upstroke of the ventricular systole, is present in the tracings from cases of "presystolic" bruit alone;<sup>1</sup> and it is inferred, either that embarrassment of the right

cate the distinction between the two sounds supposed to be produced in such close succession as to be indistinguishable by the ear.

FIG. 2.



Diagram representing varieties of presystolic bruit, copied from Professor Gairdner's 'Clinical Medicine,' p. 575 (1862).

<sup>1</sup> With two exceptions (Pl. II, figs. 13, 14), both somewhat anomalous.

side of the heart has no such marked effect in separating the auricular from the ventricular systole, which appears to be opposed to other facts, or that, when thus embarrassed, the contraction of the right auricle occurs under conditions which prevent its effect from being represented in the apex trace, such as an over-distended state of the right ventricle would necessarily present; and this is, doubtless, the more probable explanation. There are, therefore, no grounds for assigning an auricular-systolic elevation occupying the position it holds in the above cardiographic tracings to a contraction of the right auricle; if, indeed, any great embarrassment of the general circulation existed in these patients at the time when the observations were made.

This difficulty being set aside, the correspondence between the auscultatory and cardiographic indications in the above cases, would seem to amount to a proof of the fact, that the presence of mitral stenosis has no material influence in affecting the indications given by the latter of the rhythm and period of the auricular systole.

This being so, the exactly similar elevation seen in tracing *B* must indicate the occurrence of an auricular contraction of similar period to that in the two latter cases, a contraction which could under no circumstances have produced the typical presystolic bruit heard in the case from which it was taken. If capable of producing an audible sound, it would have been attended by a bruit corresponding in character with the intensification of the prolonged diastolic bruit heard in the other two cases. As no such bruit was heard in this instance, but only the bruit running up to the first sound, it may be asserted that the contraction of the left auricle here represented was incapable of producing any audible vibration in the auriculo-ventricular stream of blood. To account for this bruit as the effect of auricular contraction, it would be necessary to suppose that a second, and much more powerful, auricular contraction had occurred immediately prior to the ventricular contraction. Such an auricular contraction might conceivably be indicated in the slope at the base of the ventricular-systolic upstroke in tracings *D* and *C*; a high auricular elevation merged in that of a rapidly occurring ventricular systole, in the manner implied by the character of the bruit

itself when thus interpreted. But it is just in these cases that no "presystolic" auricular contraction occurred, as proved by the absence of any "presystolic" intensification of the prolonged diastolic bruit heard; while on the other hand, in tracing B, where, if the "presystolic" bruit heard had been really due to a second and more forcible auricular systole, this should have been very prominently indicated, no such appearance is seen.

The sloping of the lower part of the main upstroke in the above tracings probably indicates the "presystolic" commencement of the ventricular contraction, in accordance with the clinical observation of Dr. Barclay in a case of mitral stenosis where the heart was acting very slowly, in which he was able to feel a distinct cardiac movement preceding the impulse—a movement which he referred to the commencing ventricular systole on the left side of the heart, and which suggested the view advanced by him as to the causation of the bruit heard.

The evidence of these clinical facts, thus considered in their mutual relation to each other, appears to me to be strongly opposed to the "direct mitral" view of the origin of the "presystolic" bruit.

2. a. In accordance with the plan proposed, I have now to consider the alternative view of the causation of the "presystolic" bruit in cases of mitral stenosis, suggested by Dr. Barclay, namely, that the bruit is produced by regurgitation through this orifice, permitted during the commencement of the ventricular systole, but arrested at a later period by the effective closure, under the increased ventricular pressure of the valvular curtains, either merely rigid, or at the same time brought into a position less favorable for being pushed together under the force of the ventricular contraction. Such an interpretation would seem to be that most obviously suggested both by the character of the murmur itself, and by the effect which this rigid condition of the curtains must necessarily tend to produce, in interfering with the perfect action of the valve.

It is manifest, that if it were possible to hold back the mitral curtains, so as to prevent them from coming into contact during the first part of the ventricular systole, and then to let them go, all at once, in the middle of this action, a bruit of

the character of the "presystolic" bruit would be produced. Nor does it seem surprising, that the first impression conveyed by such a bruit, should have been that of its being systolic in character. By more recent writers, adopting the auricular-systolic hypothesis, this bruit has been described as not merely running up to, but as involving, or even as "appearing to take the place of,"<sup>1</sup> the first sound. A bruit so described approximates very closely indeed to the characters of a truly systolic bruit;<sup>2</sup> and it is possible that the older authorities, if not duly appreciating the diagnostic significance of the "presystolic" bruit, may yet have correctly apprehended its relation to the rhythm of the cardiac action.

But this view accords in a very complete manner with the mechanism of the mitral valve.

If the construction of this valve is considered, it will be at once obvious that the curtains can under no circumstances close without some slight reflux of blood into the auricle.<sup>3</sup>

<sup>1</sup> Dr. Sutton, 'Med. Times and Gazette,' 1866, i, p. 308.

<sup>2</sup> In the 'Archives Générales' (ser. v, vol. ii, p. 546) M. Herard relates a case in which what he describes as a systolic bruit was heard, and in which the mitral valve was found at the autopsy to be contracted, but still competent when tested by water. It is obvious, as pointed out by Marey, in reference to this very case ('Phya. de la Circul. der Sang,' p. 521), that M. Herard's position in maintaining that the bruit heard by him could not have been due to regurgitation, the valve being found to be competent, and that it must therefore, although systolic, have been produced by the direct mitral blood stream, is physically untenable. The harshness of bruit ("rude") and the constriction of the mitral orifice seem equally inconsistent with the supposition that it may have been of a "functional" character; on the other hand, the character of the bruit, as indicated by M. Herard, seems to be that of a well-marked "presystolic" bruit; and with this the fact of its occurring with the carotid pulsation, (on which account it was put down as systolic by M. Herard,) would agree, the termination of the "presystolic" bruit in the first sound and impulse being indistinguishable from the practically simultaneous carotid pulse. This is the most probable explanation of the apparently contradictory nature of the observation.

<sup>3</sup> In the 'New Manual of Physiology' (American translation of the second edition, p. 185) Professor Küss contends that the "auriculo-ventricular apparatus" must not be regarded as a valve, and that it does not in reality act as such. He supposes that the mitral and tricuspid curtains, instead of being forced into contact, and then pushed backwards into the auricles by the pressure of the blood, during the ventricular systole, are, on the contrary, drawn downwards by the papillary muscles to meet the contracting wall of the ventricle, without, in fact, coming into contact at all (see the diagrams, loc. cit.); the blood being caught

However sensitive they may be, and however readily they may be seen to "float up" toward each other in the mitral orifice under slight pressure from below, it is yet manifest that some part of the column of blood which closes the valve must run back as the curtains are brought into contact. It is maintained that the auricular pressure tends of itself directly and completely to close the valve.<sup>1</sup> A narrow stream of water directed through the mitral orifice, will indeed cause the curtains to "float up" as the ventricle becomes full, so as to occupy the aperture almost entirely; and if the stream be sufficiently forcible to distend the ventricle, the valve will close when its force is diminished or removed. In the living heart, the auricular pressure being exerted equally over the whole area of the mitral orifice, there would seem to be nothing to cause the curtains to move towards each other, so long as this is in excess of the intra-ventricular pressure, except in so far as they may be affected by the chordæ tendines as the ventricle becomes dilated.<sup>2</sup> On the reversion of the

between the approaching surfaces, and thus driven out through the aortic orifice. It is difficult, indeed, to conceive how the blood could be thus effectively "engulfed" between the mitral curtains and the ventricular walls; nor do the diagrams which are given to illustrate this view seem to render it at all more easy of comprehension. That the final completion of the emptying of the ventricle is accomplished by a drawing down of the valves, seems very probable, and would appear necessary to the absolute closure of the cavity; and this would exert that exhaustive attraction on the venous circulation to which Küas calls attention. But to assume that the auriculo-ventricular curtains do not act as true valves is opposed to the most direct inference afforded by their structure and arrangement, and to the distinct evidence, not only of daily experience in the dead-house, but of observations by Chauveau, Marey, and others, on living animals.

<sup>1</sup> 'Am. Journ. of Med. Science,' vol. xliv, p. 53.

<sup>2</sup> It is maintained by some (Brücke, 'Vorlesungen u. Physiologie,' bd. i, p. 185) that the force of the auricular ventricular blood stream, during the auricular systole, must cause the mitral curtains to be pushed towards each other by the pressure of the blood on their ventricular surfaces, which must necessarily be in excess of that of the in-flowing stream. If we supposed the mitral curtains to be hanging quite freely from their attachments, and the auricular ventricular stream to be flowing into a free space, the curtains would be brought into a position of convergence over the mitral orifice, such that they would encounter just so much of the direct impetus of the blood on their auricular surfaces as would suffice to compensate for the excess of fluid pressure on their ventricular surfaces. This last would depend upon the velocity of the blood stream, and, other things being

relation between the pressure in the two cavities, with the commencement of the ventricular contraction, the flaps will at

equal, it would vary directly with the vigour of the auricular contraction. If the ventricular pressure were raised by the influx of blood, by this, diminishing the preponderance of the auricular pressure, and hence also the velocity of the blood stream, the degree of convergence of the valvular curtains thus produced would be diminished. In any case the convergence of the curtains thus induced at the climax of the auricular systole would be lost during its decline, and could not, therefore, exert any appreciable effect in facilitating the subsequent closure of the valves, even supposing that a degree of convergence of any importance could be thus induced at all; but the actual arrangement of the mitral curtains is such that, although the smaller posterior curtain would be affected in the manner above considered, the larger anterior curtain, being held by the chordæ tendines attached to the *musculi papillares* springing from the left and posterior aspect of the ventricular wall, in a position running obliquely in front of the mitral orifice, must be thrust as far as possible from its fellow during the auricular systole, and will return to its position of rest as the force of this contraction fails. The maintenance of the curtain in such a position is manifestly most advantageous for its ready closure by the ventricular pressure, although it causes some impediment to the free entrance of blood from the auricle. It seems, then, impossible that the auricular systole can affect the closure of the mitral valve, except indirectly by distending the ventricle. Whether the mitral valve closes at the termination of the auricular systole immediately, or only with the commencement of the ventricular systole, will then simply depend upon the degree of ventricular pressure induced by the auricular systole; whether this is greater than the force of the blood discharged into the auricle as its walls become relaxed; it may be, according as the essential object of this rapid contraction of the auricle is to effect a forcible, or a rapid, filling of the ventricle, to stimulate it to contract, or merely to help the circulation over the "dead point" of its course, and to do this with as little impediment to the pulmonary circulation as possible. The loudness of the first sound shows that the ventricular pressure is not great at the commencement of the systole, and that the muscle has that freedom at the commencement of its contraction which is necessary for its effective action. This would be afforded by the bulging upwards of the valve into the auricle after the edges of the curtains have come into contact. As this occurs, the mitral curtains and the wall of the ventricle are simultaneously rendered tense, and thrown into those active vibrations to which the first sound is mainly due. If the thick fleshy mass of the latter is compared with the membranous character of the former, and if it is considered that, while the valvular curtains are in contact with a fluid medium on both sides, the ventricular wall is partly in contact with the thoracic wall, partly with the lung and other thoracic organs, and on one side attached to its fellow organ, it can scarcely be doubted that the loudness of the first sound is due chiefly to the valvular vibrations, and this is entirely corroborated by the repeated experiments of Chauveau and others, in which these valvular vibrations were felt by the finger, and in which it was further found that, when these were arrested by the introduction of the finger into the mitral orifice, the first sound ceased to be distinctly heard.

once be pushed together by the pressure from below preponderating. I am inclined to attribute the slight prolongation of the normal first sound to the vibration of the membranous curtains produced by the slight reflux necessarily attending the closure of the valves in this manner, rather than to muscular susurru, or to fluid or parietal friction of the blood. Sounds due to these latter causes must continue throughout the whole period of ventricular systole, and if audible would be heard as a soft and continuous systolic murmur.

It thus appears to me that in the normal first sound of the heart the rudiment of a "presystolic" regurgitant bruit is heard.<sup>1</sup> If there should be a slight thickening, causing some loss of sensitiveness in the mitral curtains, or perhaps if there should merely be a sluggishness in the ventricular contraction, there would result an increase of this "presystolic" regurgitation, and a corresponding increase of this vibratory element of the first sound would be heard as a slight prolongation and roughening of it. Where, as in an advanced case of mitral stenosis, the mitral curtains are obviously thickened and stiff, it can scarcely be imagined that their closure could be effected without considerable reflux, sufficient to produce a well-marked "presystolic" bruit. A delay of the closure of the valve thus caused would at the same time result in a more forcible collision of the curtains, which may account for the loudness and sharpness of the first sound heard following this bruit.

If the mitral curtains should become more contracted, so as to be no longer capable of being brought into exact apposition, the harsh rough "presystolic" bruit would be followed by a softer blowing murmur of continued regurgitation; the softer character of the latter being due to the fact that the contact of the mitral curtains at their free edges must check the vibrations readily produced in them by the more free reflux previously permitted, vibrations on which the loudness and harshness of the "presystolic" bruit would seem to depend. The "post-systolic" (or systolic) portion of the bruit, being thus

<sup>1</sup> The contrast between the "*tic-tac*" of the foetal heart, and the "*lub-dub*" of the normal cardiac sounds, is thus satisfactorily accounted for, if referable to an extreme degree of delicacy and sensitiveness of the foetal valves; but not so well, if attributed to an absence of hypothetical muscular or frictional elements of the first sound.

more or less due to the fluid vibration of the blood returning into the auricle, would be softer the more fully in contact the curtains are brought, and *vice versa*. If, finally, a retracted state of the mitral valve should be arrived at, in which the flaps are incapable of being brought into contact at all, a uniform character of the bruit would be maintained throughout. Under such conditions, however, greatly increased thickening and rigidity of the curtains might at the same time diminish the harsh quality of the bruit by interfering with their vibratory qualities.

Clinically, we meet with auscultatory phenomena corresponding with all those here indicated, the purely "presystolic" bruit of all degrees from the well-marked typical bruit down to what can scarcely be considered as more than a modification of the first sound;<sup>1</sup> the "presystolic" bruit followed by a distinctly systolic bruit of varying character and intensity; and finally a continuous systolic bruit, distinctly commencing before the impulse, and without any appreciable alteration in its character. The above explanation of these phenomena seems at least as satisfactory as that given of them on the auricular-systolic hypothesis.

The above considerations at the same time show clearly, that the condition of the mitral valve, present in these cases, must necessarily have tended to produce just such effects, in a

<sup>1</sup> A modification of the first sound, corresponding with a description which I have happened to meet with in a clinical note made by Dr. Sutton, in a case of spinal cord disease, with simple hypertrophy of the left ventricle. The sound was peculiar, being "a low-pitched sound followed by an abrupt knock, the apex beat against the chest." The first part of the prolonged sound was regarded as "presumably due to contraction of the muscle, which is not generally very distinct, here very distinct indeed; seemingly the healthy sounds all exaggerated." In regard to this observation Dr. Sutton tells me that a modification of the first sound of this kind is very common, and one with which he is very familiar, but one which is not associated with any structural lesion of the valves. Such a prolonged, somewhat rough, and abruptly terminated first sound I am continually meeting with in the out-patient room. In such cases I regard it as indicating an imperfect action of the mitral valves from slight thickening of the edges of the curtains, with some weakness or irregularity of the cardiac action, or possibly from the latter cause alone. I cannot, for reasons above indicated, agree in attributing it to muscular contraction. In the case now referred to the autopsy showed that there was no obvious disease of the valves of the heart.

greater or less degree, in the manner now contemplated ; and it can only have failed to do so, if this tendency had been counteracted by a simultaneously and proportionately increased vigour of the ventricular contraction. It was pointed out, however, that there is no evidence of this being brought about ; but that, on the contrary, the slowness of the cardiac action observed in cases of mitral stenosis, and the effect of this in diminishing the work required of the ventricle, concur in showing, that the vigour of its action tends to become less instead of greater, in a manner calculated to bring out prominently the effect of the rigid mitral curtains in producing a "presystolic" regurgitant bruit.

From this point of view, the occurrence of a "presystolic" bruit in the presence of rigid mitral curtains, and with a slowly acting ventricle, seems to admit of no other physical explanation than that advanced by Dr. Barclay.

Dr. Barclay's view, if thus in accordance with the various character of bruits heard in cases of mitral obstruction, is supported by observations which directly indicate the occurrence, in some of these cases, of appreciable "presystolic" ventricular movements.

Dr. Barclay himself, in the memoir referred to,<sup>1</sup> mentions the fact that, in one of the cases recorded by him, he was able to perceive a movement of the heart distinctly preceding the impulse—a movement which he considered attributable to the commencement of the ventricular systole alone. The accuracy of this observation, and the justness of the inference drawn from it, are corroborated by the evidence of the more delicate indications of the cardiograph. When discussing the bearing of Dr. Galabin's cardiographic tracings on the auriculo-systolic hypothesis, I observed that the gradual rise seen at the base of the upstroke in tracing *b*, slightly indicated in tracing *c*, could not be referred to a second auricular contraction, because the bruit, instead of being again intensified, as it would then have been, disappeared before the first sound ; and the inference was drawn that this must really indicate the commencement of the ventricular contraction.<sup>2</sup>

<sup>1</sup> 'Lancet,' 1872.

<sup>2</sup> This may, however, have been due to the contraction of the right ventricle having commenced before, or having been felt before, that of the left ventricle.

The above facts connected with the occurrence of the "presystolic" bruit in cases of mitral stenosis seem to me to show—

1. That the auricular-systolic hypothesis implies the occurrence of certain modifications in the character and relations of the auricular and ventricular contractions, of which no distinct evidence is adducible, and which appear incompatible with the evidence afforded by cardiographic and auscultatory observations made in these cases.

2. And, on the other hand, that the character of the "presystolic" bruit, and the complications it presents, are those of a bruit which would necessarily be produced, in a greater or less degree, under the conditions presented by a rigid state of the mitral valves, and which would be developed in a proportionately prominent manner by a retardation of the activity of the ventricular contraction, such as a narrowing of the mitral orifice is found to produce.

## *II. Cases in which a "presystolic" bruit had been heard during life, but where the autopsy has shown that no appreciable contraction of the mitral orifice had existed.*

I have now to pass on to the consideration of certain cases, in which this peculiar bruit has been heard in the absence of narrowing of the mitral aperture.

It is obvious that, if a "presystolic" bruit is truly an indication of partial incompetence of the mitral valve, it should be met with under any conditions tending to affect the efficiency of the valve, as in the case of dilatation of the cavity of the ventricle. That a bruit of this character is sometimes heard under such conditions is shown by the observations made in the cases which I have now to bring forward—cases in which a bruit of typical "presystolic" character was heard during life, but in which post-mortem examination showed that no contraction of the mitral orifice had existed.

Two such cases are recorded by Dr. Austen Flint, in vol. 44 of 'The American Journal of Medical Science.'

Case 1 is that of a patient, æt. 56, with aortic regurgitation, and dilatation and hypertrophy of the heart, who suffered from repeated attacks of an anginose character, in one of which he finally died. When the patient was examined during life a "presystolic blubbering" murmur was heard at the apex, and a regurgitant aortic murmur at the base, audible over nearly the whole praecordium. There was no systolic bruit either at apex or base. The "presystolic" bruit" was of the character which Dr. Flint "then supposed to be characteristic of the button-hole mitral orifice." After death the heart was found to be hypertrophied, weighing sixteen ounces and a half, and the wall of the left ventricle measuring four fifths of an inch in thickness. "The mitral valve presented nothing abnormal save a few small vegetations at the base of the curtains as seen from the auricular aspect of the orifice." The aortic valve was incompetent.

In Case 2 there was also aortic disease, a double murmur being heard at the base. "There was also a distinct presystolic murmur within the apex, having the blubbering character." On examination post-mortem "the mitral curtains presented no lesions; the mitral orifice was neither contracted nor dilated, and the valve was evidently sufficient. The heart was considerably hypertrophied, weighing seventeen ounces and a half, and the wall of the left ventricle was an inch in thickness." The aortic valve was insufficient.

In regard to these cases Dr. Flint observes, "In both cases the mitral direct murmur was loud, and had the character of sound which I suppose to be due to vibration of the mitral curtains." He considers that these cases are capable of being satisfactorily explained in accordance with the auricular-systolic hypothesis, here adopted by him. He refers to experiments made by Drs. Baumgarten and Hamernik, who showed that a forcible injection of fluid into the ventricle through the mitral orifice will cause complete closure of the valves by coaptation of the curtains, these being "floated out" and brought into apposition by simply distending the ventricular cavity. He then observes that, "with aortic incompetence," present in both the above cases, "the left ventricle is rapidly filled with blood before the auricular systole takes place. This dilatation is such that the curtains are brought into

coaptation, and when the auricular systole occurs, the mitral direct current passing between the curtains throws them into vibration ; the physical condition being in effect analogous to adhesion of the curtains at their edges, the condition under which this bruit is ordinarily produced."

The experimental results here referred to will be readily accepted, for the closure of the mitral valves by distension of the ventricles, is virtually the same thing as its closure by ventricular contraction, the fluid pressure being the efficient cause in both cases. But there seems to me to be much difficulty in accepting the views of Dr. Flint based upon inferences drawn from these observations. Supposing that mitral curtains had been already brought into contact by the ventricular pressure when the auricle was about to contract, they would yet have been incapable of offering any obstacle to the entrance of blood, so soon as the auricular should have exceeded the ventricular pressure ; and they would have been pushed apart to an extent proportionate to the preponderance of the former over the latter pressure. If the auricular pressure had been only just in excess, but a few drops would have entered the ventricle through a narrow chink ; under a higher pressure a larger volume of blood would have entered through a wider opening. There could never under such circumstances have been a rapid and forcible influx of blood, such as would have been necessary to the production of a direct mitral murmur of a pronounced character. And moreover, the distended condition of the ventricle is even more distinctly incompatible with the production of such a bruit, which especially demands the presence of a comparatively free space in front of the seat of obstruction. When Dr. Hope produced a systolic bruit over the aorta by successive bleeding of an animal, this result was probably due as much to the resulting diminution of the arterial pressure encountered by the blood issuing from the ventricle, as to the lessening of its viscosity by the induction of a condition of more or less hydæmia.

In regarding the bruit heard in these cases of Dr. Flint's as due to mitral regurgitation, no such difficulty is encountered. The facts so viewed admit of very simple and natural explanation. For though it seems difficult to understand how

ventricular dilatation can lead to complete mitral insufficiency, the manner in which it must tend to produce a partial failure of its efficiency, such as the occurrence of a "presystolic" bruit would indicate on this hypothesis, is readily intelligible. If owing to ventricular dilatation the insertions of the musculi papillares should be so far removed from the mitral orifice as to prevent the curtains from meeting at the commencement of the ventricular systole, they may yet meet when a certain amount of contraction of the cavity is accomplished, and in this way a regurgitant "presystolic" murmur might be produced. It would seem to be almost a necessity that such an intermediate state should intervene between competence and complete insufficiency of the mitral valve produced by simple dilatation. When, however, the valvular failure depends upon a yielding and inversion of the edges of the valves themselves, such an intermediate stage would not occur. This may in part account for the fact that the occurrence of the intermediate stage of partial regurgitation is so rarely observed. Two other considerations seem to me to tend in some degree to explain what would appear to be so anomalous:—1, that it is probably only the complete failure of the protection to the pulmonary circulation afforded by the mitral valve, which determines the supervention of those more serious results of cardiac failure which bring the patient under observation; and, 2, that possibly some at least of the cases of "presystolic" bruit put down as cases of mitral constriction, may really be cases in which dilatation of the ventricle, or other cause, may have led to a partial incompetence of the valve without stenosis. It is just these cases that are least likely to afford opportunities of verification of the clinical diagnosis, unless it be in a manner accidentally. In two of the cases now to be mentioned as having come under my observation, such a false impression was received, and was corrected by the revelations of the autopsy, the patients having neither of them died from the effect of the valvular failure. I am inclined to believe, however, that this commencing, or perhaps merely temporary, mitral failure is often indicated by a slight roughening of the beginning of the first sound only, an indication which, though slight, seems sufficiently distinctive in character when attentively observed. My impression is,

from my own experience, that a slight "presystolic" bruit is of exceedingly common occurrence.

However this may be, the absence of such cases as these described by Dr. Flint would appear to me to be a thing more difficult to account for than their apparent rarity.

The cases observed in the wards of the hospital presented characters, which seem still less in accordance with the auricular-systolic hypothesis, than those recorded by Dr. Flint.

I am indebted to Dr. Peacock and to Dr. Bristowe for permission to make use of the two following cases. The particulars of the cases I have taken from case-books and papers in the wards, and have introduced notes made independently by myself at the bedside, in reference to the cardiac phenomena especially.

*CASE 1. Diseases of the aortic valves; dilated and hypertrophied left ventricle; a "presystolic" alternating with a systolic bruit heard at the apex of the heart; dilatation of the mitral orifices; no disease of the valves.*—Henry Charles H—, æt. 33, was admitted into St. Thomas's Hospital under the care of Dr. Peacock, March 29th, 1876.

He had been in the army, and had served thirteen years in India. He had received his discharge fourteen months previously on account of failing health; since that time he had been employed as a waiter.

His father died suddenly from heart disease at the age of fifty-seven; his mother was then still living at the age of seventy-two, suffering from bronchitis and "asthma." His brothers and sisters, with one exception, were also alive and in good health.

When ten years old he had scarlatina, which was followed by dropsy. He stated that he had never had rheumatism; he had been ailing for two years, but had not been laid up until about two months before his admission into St. Thomas's. He was then (January, 1876) admitted into the London Hospital with anasarca. He improved very much and was sent to Woodford; while there, however, he caught a fresh cold, and had been gradually getting worse, with a return of his former symptoms.

At the time of his admission into St. Thomas's Hospital

he had a puffy expression of countenance with flushed cheeks, and a general icteroid tinge of the skin. He had a frequent cough, hurried breathing, and was expectorating a considerable quantity of muco-purulent sputum tinged with blood. The chest was fairly resonant in front, somewhat dull posteriorly, more particularly in the dorsal region, where also the breathing was somewhat feeble and attended with rhonchus. The praecordial region was somewhat prominent; the area of dulness increased, commencing above in the second interspace, and extending laterally from the left side of the sternum to a finger's breadth within the nipple. There was a double murmur heard at the base, and over a large portion of the heart, of which the diastolic portion was the louder, harsher and more prolonged. This was perhaps audible more distinctly and to a lower point on the left side of the sternum, but was very distinct on the right side of the sternum also. The liver extended high into the thorax, and about an inch down into the abdomen; it gave a sense of hardness in the epigastrium, and was tender when percussed. There was a general tremulous movement in the praecordial region, but the point of pulsation of the apex could not be felt. The tongue was covered with a thick white fur. The urine was diminished in quantity, and contained "one sixth" of albumen; sp. gr. 1026.

On April 3rd I made the following note as to the condition of the heart. The heart's apex seems to be beating under the sixth rib, two inches below the nipple and about in the mammary line. The impulse is diffused somewhat; the impulses are stronger and weaker alternately, the former being attended with a slight thrill. On auscultation a systolic and a presystolic bruit are alternately heard, the latter attended by a sharp first sound; after each a diastolic bruit is heard, conveyed from the base, and a carotid pulsation accompanies each.

The following notes, dated April 4th, are taken from Dr. Peacock's case-book:—"He has been upon the whole better. There is a very peculiar double murmur heard at the base of the heart, which sounds extremely superficial, and of which both portions are very short, and do not seem distinctly to accompany either the systole or diastole entirely,

though on advancing down the sternum it seems to lapse into an ordinary diastolic murmur. Towards the apex, a little to the left, there is an abrupt murmur, which seems to precede the systole; but if so it is much longer than an ordinary presystolic murmur; it certainly terminates with the impulse of the heart. The apex appears to beat in the fifth interspace, about one inch and a half below the nipple, and in a line with it, and there is there a slight thrill. The murmur is not heard behind. The pulse in both wrists has a distinctly regurgitant character. At the upper part of the sternum on the right side the double murmur is very distinct. The dulness upon the top of the sternum is perhaps slightly increased."

I have another note made on April 6th:—"The heart's action is very irregular, stronger beats intermingled with weaker ones. The double bruit at the base is rough; at the apex a diastolic bruit is heard with each beat, possibly conducted from the base, but softer and more uniformly prolonged; accompanied by a thrill at the apex, every third beat or so, a distinct presystolic bruit is heard with a sharply accentuated first sound; with other beats the bruit here is systolic."

I made a further note on April 8th:—"A systolic and a 'presystolic' bruit are heard alternating with each other, a diastolic bruit is heard after each, and each is accompanied by a distinct carotid pulsation. The presystolic bruit seems to be attended by a stronger cardiac impulse. The first sound seems to be lost with the systolic bruit. There is a thrill at the apex, which seems to be more distinct with the stronger impulses; it seems also to be diastolic: it is less marked than before."

Similar physical signs in connection with the heart continued to be heard with slight variations for some time.

A further note occurs in the case-book, dated April 21st, to the following effect:—"His appearance has much improved since his admission, the jaundiced tinge having much subsided. The pulse is to a much less remarkable degree regurgitant, it is occasionally intermittent and somewhat irregular. The praecordial dulness does not rise to a higher level than natural, but is very much extended to the left. It is, however,

impossible to say how far it extends in this direction, as there is some effusion into the left pleural cavity, indicated by entire dulness on percussion, and absence of vocal thrill and respiratory sounds. The point of pulsation of the apex of the heart cannot be felt; but it is probably in the fifth interspace, within the line of the nipple and about two inches from that point. The heart sounds are much more marked than before, but the double murmur at the apex is distinct, and there is occasionally a very superficial systolic sound. At the apex there is perhaps a presystolic murmur."

It is noted also that the urine, which by the 13th had become abundant under the use of digitalis, and had ceased to give any indication of the presence of albumen, had again diminished in quantity to 25 oz. in twenty-four hours, when a mixture containing quinine and iron was substituted for the diuretic, and this had again increased in quantity since a return had been made to the digitalis on the 17th.

April 25th it is noted that the patient was not so well. The jaundice was marked, and albumen had again appeared in the urine, the quantity being reduced to a pint. A distinct double murmur was heard at the base, and a systolic murmur at the apex; the superficial sound was not heard. The patient was suffering from vomiting, and he died somewhat suddenly in the course of the day.

The following are Dr. Peacock's notes of the condition of the heart after removal:

The girth of the right ventricle	=	6 inches, 8 lines,
"        left        "	=	6        "
The pulmonic orifice	=	3.72 " (No. 11),
"        aortic        "	=	3.72 "        "
Right auricular ventricle aperture	=	4.79 " (No. 15),
Left        "        "        "        "	=	4.79 "        "
Walls of right ventricle	measure	2, 1½, 1 lines,
"        left        "        "		5, 5, 3        "

The aortic valve is very incompetent, very shallow, especially the right and posterior segments; there is a band of lymph on the lining membrane at the base of the ventricle, at the angle of attachment of the right and posterior segments, and there is some also near the mitral valve. The

mitral valve is much thickened; the ascending aorta is much dilated, and its coats are thickened. The weight of the heart is 20½ oz. There are white patches on the external surface of the right ventricle. The lining membrane of the left auricle is very thick and opaque. When recent some old loose fibrous bands were seen about the base of the heart and around the aorta and pulmonary artery. The mitral valve was found to be competent when tried by the water test.

In the above case the notes, taken from Dr. Peacock's case-book, in which they were written down at his dictation, distinctly indicate the existence of an abrupt bruit preceding the first sound, and terminating with it. Examining the case more frequently, and perhaps sometimes under more favorable conditions, I could feel no doubt as to the distinct "presystolic" character of the bruit heard. In this case the circumference of the left auriculo-ventricular aperture was greater than normal. For the notes of the next case I am chiefly indebted to my friend Dr. Twining.

*CASE 2. Disease of aortic valve; dilatation and hypertrophy of the left ventricle; systolic and presystolic apex murmur.*—Thomas P—, æt. 36, a draper, was admitted into Charity ward under the care of Dr. Bristowe, on July 6th, 1876, suffering with symptoms of *morbus cordis*.

He stated that, with the exception of occasional "bilious attacks," he had enjoyed good health until four months before. He had never had rheumatism nor gout, nor was there any history of either disease in his family. His father had lived to the age of seventy, and his mother was still living at that age; he had three brothers and a sister alive, and one sister had died. He denied having suffered from venereal disease. Four months before admission he began to feel a pain across his chest at the end of the day's work, but it was not until two months later that he first began to think there was anything amiss with him. He then consulted a doctor, because his wife said that he was getting stouter. Since this time he has found his breath short after walking, especially up hill. His legs began to swell six weeks ago, and this swelling has progressed since. He has had no cough nor expectoration.

On admission the patient was pale, and so short of breath as to be unable to lie down; the back and legs, and especially the thighs, were oedematous. The pulse was very irregular, about 108, one strong beat alternating with two feeble ones, with occasional intermissions. The tongue clean and the appetite good. He said that for two weeks he had slept badly; he had not been troubled with dreams, nor with headache. On examining the chest the following condition was found by Dr. Twining:—The apex of the heart was beating two inches outside the nipple and one inch below its level. The action of the heart was too irregular for satisfactory auscultation, but there seemed to be a bruit synchronous with the pulse, audible about the second left interspace, and mid-sternum. The lungs were resonant; occasional dry râles were heard over the chest generally, except at the bases where moist crepitations were heard. The hepatic region was tender to percussion, the tenderness extending below the ribs.

The following note was made by Dr. Bristowe a day or two later:—"A diastolic aortic bruit is audible mainly at the base and along the sternum. There is an indistinct systolic murmur at the base chiefly audible to the left of the sternum, and a doubtful systolic murmur at the apex. The action of the heart is sometimes regular and sometimes intermittent. The apex beats an inch to the south-east of nipple."

At a later date a diagram was made by Dr. Bristowe on the patient's bed card, representing a diastolic followed by a "presystolic" bruit, but no note accompanies it.

I have the following note made on July 10th:—"The heart's action unequal. A prolonged diastolic bruit is audible at the apex, as well as a systolic bruit without audible first sound; but with the stronger beats the first sound is sharp and flapping, and preceded by a presystolic rough bruit; nothing is heard following it. From time to time with the weaker beats there is a bruit of a presystolic character. The presystolic bruit coincides with the carotid pulse; there is a marked thrill at the apex, most distinct with the stronger impulses, apparently when the presystolic bruit is most distinct."

On July 11th Dr. Twining's note is, that the patient's

breathing was less easy, and that the œdema had increased, and had affected the scrotum.

On July 12th I made a further note, to the effect that the systolic bruit was loud at the end of the sternum ; that at the apex a diastolic and a presystolic bruit were audible, the diastolic bruit at the base being apparently distinct from the other. Venous pulsation was visible in the neck.

I had no further opportunity of examining the patient. His condition got gradually worse. Petechiae appeared over his back and legs ; he became jaundiced, and finally died on August 15th.

On post-mortem examination both ventricles of the heart were found to be dilated and hypertrophied, and both auricles dilated. The right auriculo-ventricular orifice admitted five, and the left three fingers. The curtains of the mitral valve were thickened and rough at their margins, but apparently competent. The aortic valve was thickened, but no patency of it, or of the pulmonary artery, was observed. The aorta was dilated, no appearance of atheroma. There was commencing fatty degeneration of the muscular tissue at the base of the left ventricle, and in the musculi papillares.

In this case, although there is no reference in the notes of the case to the little diagram appended by Dr. Bristowe, representing, as before said, a diastolic followed by a "presystolic" bruit running up to the first sound, I have his authority for saying, that he has no doubt whatever that he heard the bruit which it is intended to represent, though he has no distinct recollection of the observation at the present time.

In neither of these cases was there anything to indicate structural incompetence in the appearance of the mitral curtains, and in Case 1 they were shown to be competent by being tested. Hence the occurrence of regurgitation can scarcely be explained on any other hypothesis than that they were prevented from meeting either by the effect of dilatation of the ventricle, or by irregular action of the musculi papillares. There was no indication of the edges of the curtains having been everted by the ventricular pressure. Supposing the valve to have been held open by mere distension, or by

the papillary muscles, the contraction of the ventricle more or less nearly to its full extent, would have permitted them to come into contact at a later period of the systole, and a regurgitant "presystolic" bruit would under such circumstances have been produced. If the ventricular systole, however, had been feeble, or commenced from a condition of great distension, and incomplete, its vigour might have failed before the cavity was sufficiently reduced to allow the mitral curtains to come into contact, or it might have been too feeble to bring the somewhat thickened curtains efficiently into apposition; a continuous systolic bruit would then have been audible, with an absence, or enfeeblement of the mitral "systole." Such an explanation of the direct effect of ventricular dilatation, present in both the above cases, agrees very closely with the clinical phenomena observed in connection with them, and is that which they themselves suggested.

The two following cases, the first of which was a comparatively short period under observation, attracted less special attention, the "presystolic" bruit occasionally audible being regarded as indicative of some contraction of the mitral valve. The termination of each case occurred independently of the valvular lesion, and hence they are of interest as having incidentally afforded means of verifying the fact of the occurrence of a "presystolic" bruit, under conditions in which it has been regarded as indicative of mitral obstruction, when in reality attributable to the ventricular dilatation.

I am indebted to Dr. Stone for kindly permitting me to introduce them here.

**CASE 3.** *Syphilitic disease of the liver; lardaceous disease of kidney, spleen, and intestine; a variable systolic and presystolic bruit at the apex of the heart; no valvular lesion found.*—Sarah R.—, æt. 35, a widow, in service, was admitted into St. Thomas's Hospital under the care of Dr. Stone, on April 19th, 1876.

When twenty-one she had an attack of acute rheumatism for the first time, and she had a second attack when twenty-nine, in which "her heart was affected." After this attack she had some anasarca, which, however, passed off in a short

time. Ever since this time she was subject to palpitation. For twelve months before her admission she had found her breath getting short, and her hands and feet cold; she had had no faintings. During three months she had been troubled with cough, and had noticed swelling of her ankles, which had gradually extended up the legs. She had had no haemoptysis. She stated that she had been subject to a winter cough, but it had been much worse during the previous winter. The catamenia had been absent for five months.

On admission the patient was exceedingly weak and prostrate; there was considerable oedema of the legs, and some ascites. Her urine had a specific gravity of 1010, and contained much albumen ("one third"). It was noted that the apex of the heart beat half an inch below the nipple, and that the impulse was accompanied by a thrill, and that a systolic bruit was audible.

On April 22nd I noted—The heart's apex beats just below and inside the nipple; there is a distinct thrill attending the impulse and terminating with it; here also there is a short bruit running up to the first sound.

On the following day Dr. Stone noted that the heart's action was visible and undulatory, with retraction of the intercostal spaces, and attended by a thrill; the first sound ringing and reduplicated, murmur (?).

On April 26th I noted that there was a short presystolic bruit; no systolic bruit.

On May 6th I made the following note about the condition of the heart:—There is a marked thrill at the apex with the impulse; there is a slight systolic bruit audible at the apex only, it is not abruptly terminated; the second sound is prolonged over the third costal cartilage.

This patient got gradually weaker, and died on May 9th, her death being accelerated by an attack of diarrhoea.

On post-mortem examination it was found that the heart, though somewhat enlarged symmetrically, was perfectly normal in respect to the condition of its orifices and valves, except a small patch of atheroma on the anterior flap of the mitral, adjacent to the aortic valve. The mitral orifice measured four inches and one-third in circumference. Very slight atheroma of arch of aorta; a few raised, gelatinous patches of

endarteritis in the abdominal aorta. The liver was deeply fissured and contained gummata; the kidneys, spleen, and the mucous membrane of the intestine were lardaceous, and there were other indications of syphilis.

This patient I supposed to have some mitral stenosis, attended with a short, but typical, and characteristically variable bruit. This view accorded with her state of prostration and cold extremities. The normal condition of the mitral valves found at the autopsy, gave the case an especial interest which it had not seemed to present before.

The systolic character of the bruit when the patient was first admitted, its assumption of a "presystolic" character shortly after, when the condition was somewhat improved, and its again becoming systolic shortly before death, may properly be compared with the variation of the bruit heard in the two preceding cases, occurring successively with the stronger or less embarrassed, and with the less effective ventricular contractions. The degree of failure of the valve in this instance would seem to have depended upon the degree to which its weakened walls yielded to the pressure of the blood, together with a concurrent enfeeblement of its action. In accordance with this, there would seem to have been a slight return of the apex beat towards its normal position, concurrently with the appearance of the "presystolic" bruit.

In the following case, a possible element of uncertainty might be thought to occur, in the presence of pericarditis at the autopsy. This, however, was of quite recent date, and is to be regarded as the final cause of the fatal issue. It seems scarcely possible, that it can have had anything to do with the physical signs, presented by the heart between five and six weeks before, at a time when the patient's temperature was observed to be normal. In any case it may be said, that the "presystolic" bruit is less likely than any other endocardial sound, to be simulated by pericardial friction. It is indeed scarcely conceivable that a friction sound, due to the presence of recently formed lymph at the apex of heart, should be abruptly arrested at the moment of the first sound, which marks the commencement, and not the conclusion of the stroke of the heart by which the friction sound is produced; though such a sound might possibly be caused by the

tightening of old fibrous bands connecting the pericardial surfaces.

**CASE 4.—***Thickening and incompetence of the mitral valve; dilatation and hypertrophy of both ventricles; a soft diastolic, a "presystolic," and a systolic bruit at the apex.*—Emily S—, aged 32, widow, working as a charwoman, was admitted into St. Thomas's Hospital under the care of Dr. Stone, on June 3rd, 1876.

She said that three years previously she had had an attack of rheumatic fever for the first time, which laid her up for twelve weeks. She did not find her breath short after this illness. Twelve months before her admission she had a second attack of acute rheumatism, for which she was treated at the Middlesex Hospital, where she remained three months and a half. During this attack she had precordial pains, with palpitation and cough; she had poultices applied to the chest, and subsequently belladonna plaster. Ever since this time she had been weak, and her breath had been short. During the last five weeks she had been getting much worse, with increasing shortness of breath, cough, and vomiting. For the last fortnight she had noticed that she was becoming jaundiced, and she had finally been laid up a week before her admission with swelling of the legs.

With regard to her family history, her father, one brother, and three sisters were all strong and healthy; her mother had died of "consumption" at the age of thirty-two; she had also been the subject of a hernia; none of them had been subject to rheumatism or "gout" or had had rheumatic fever. She herself had always been in good health up to the time of her first attack of acute rheumatism. She had had four children born alive and one miscarriage: one child had died at the age of two years and a half of "consumption."

On her admission she had oedema of the legs, her face was puffed and flushed, and slightly jaundiced. The temperature was slightly raised,  $100^{\circ}6$  in the evening, but fell to  $98^{\circ}$  on the following day and continued normal. The pulse was 112 and the respiration 32. There were then no signs of rheumatism. On examining the chest a systolic bruit was heard at the apex, and the heart appeared to be hypertrophied, the

transverse area of dulness being increased to four inches. The liver dulness was also increased and the organ tender.

Examined on June 8th, the urine was found to have a sp. gr. 1028, and to contain a trace of albumen. On the 16th this no longer appeared, and the sp. gr. was 1018.

Dr. Stone's note on June 19th was, that a double murmur was audible at the apex and also some prolongation of the first sound to the right of the sternum.

On June 21st I made the following note of the condition of the heart:—The cardiac impulse is distinct, not heaving in character; it is felt under the fifth rib, in the line of the nipple. At this point a double bruit is heard, a soft prolonged diastolic bruit, commencing soon after or immediately following the second sound, increasing in loudness and falling again, and almost continuous with a systolic bruit which runs up to and through the impulse, the first sound being well marked and accompanying the impulse. The commencement of this bruit is of the character of a "presystolic" bruit. A systolic bruit is audible behind on both sides of the chest. The sounds at the base are clear. There are signs of congestion of the bases of both lungs. The liver is enlarged and firm.

On July 1st I made the short note:—The systolic murmur at apex is alone distinct, diastolic bruit (?)

I have another note made on July 5th:—Inside the apex (situated as before) a diastolic and a systolic bruit are heard, both soft, the systolic bruit commencing before the impulse. At the apex itself the diastolic bruit is heard, with a "presystolic" bruit running up to a sharp first sound, and followed by a short systolic bruit, scarcely audible. There is a slight diastolic thrill at apex. This patient died on July 15th with acute pericarditis.

On post-mortem examination the following condition of the heart was found:—The organ was enlarged, weighing twenty ounces; the surface was covered with recent lymph; there was dilatation and hypertrophy of both ventricles, the walls being soft and flabby. The aortic valve was diseased, and apparently incompetent, the mitral valve also was thickened, and incompetent. The lungs were œdematos, and some old adhesions existed over part of the left lung. The liver weighed

two pounds, and had a nutmeg appearance. The spleen weighed six ounces, was firm and congested. The kidneys were pale on the surface; they weighed thirteen ounces.

The impression received in this, as in the preceding case, was that, with the manifest incompetence, there was also constriction of the mitral valve; and in this case, too, the results of the autopsy were unexpected. The diseased condition of the aortic valve, and the suspicion of its incompetence, might suggest the possibility of the diastolic and "presystolic" bruits heard at the apex, having been an aortic regurgitant murmur. But the character and situation of the bruit are quite inconsistent with such a supposition. In this case the thickening of the mitral curtains, and the dilatation of the left ventricle, are both elements tending to the production of a "presystolic" regurgitant murmur. The soft diastolic apex bruit was such as is not unfrequently heard where no narrowing of the mitral orifice exists, and sometimes where there is no roughening of the valve.

The six cases here mentioned tend to invalidate in some degree, the precise significance at present attached to the "presystolic" bruit. As already pointed out, I am unable to conceive of any other explanation of the occurrence of this bruit, in the absence of mitral obstruction, than as the result of regurgitation.

### III. *Cases in which a bruit has been heard similarly related to the second sound, and having a character identical with that of the "presystolic" bruit.*

There is one more clinical fact, which gives very direct and material support to this same view of the question. I refer to the occasional occurrence of a distinct bruit at the base of the heart, of a character identical with that of the "presystolic" bruit, and related in a precisely similar manner to the second sound, as this last is to the first sound of the heart.

In some of our text-books but little reference is made to the occurrence of this "prediastolic" bruit. Dr. Walshe, however, enters into some discussion on the subject. Speaking of the diastolic basic bruit, he says, "The second sound may

be covered completely at the maximum of the murmur, or it may occur at the beginning of, during, or at the close of this. In the first class of cases the valves are utterly incompetent. In the varieties of the second class of cases one division of the valve may flap naturally, or all three imperfectly, so as to allow of the production of an imperfect second sound. The most common of these compound conditions of bruit and sound at the base is that of a murmur abruptly brought to a close by the second sound; a state pretty accurately represented by the whispered symbols *phwi . . . tt*, *phwe . . . tt*, the "*tt*" being sharply accentuated." He further observes that "in such cases the explanation may be that *partial* aortic reflux takes place, the valve being partially, not wholly disabled from closing." He suggests the possibility of the effect being due to a short regurgitant murmur at the aortic orifice followed by a delayed pulmonary sound; "but," he goes on to say, "the impression on the ear is so strong of the two divisions of the *phwi . . . tt* sound being produced at the same spot, and being distinctly connected with each other, that it is difficult to accept this interpretation."<sup>1</sup> It is also difficult to imagine that in the case of complete aortic incompetency, where the valve is incapable of being effectively closed at all, so short a bruit could occur as to be capped by a delayed pulmonary sound.

A temporary reflux, owing to a delay in the closure of the valve, is the obvious, and apparently the only intelligible explanation of the "prediastolic" bruit, whether produced in the aorta or pulmonary artery. From the construction of the semilunar valves, as in the case of the auricular ventricular valves, it is manifest that, in the normal and most perfect closure of the valve, some slight reflux must occur. And in the case of these valves also, if a condition of the valvular curtains occurs capable of retarding their meeting for a sufficient interval, an audible bruit will be produced, abruptly terminated by the sharp click of their sudden impact, accentuated in proportion to this delay. I believe that a slight bruit of this character is not very uncommonly to be heard preceding the second sound—what might be described as a modification, or roughening of the sound, but distinctive in

<sup>1</sup> Dr. Walshe, 'Diseases of the Heart,' 4th edition, p. 103.

its character, and as such indicating a slight impairment of complete efficiency of the aortic or pulmonic valve. In the two following cases a distinct "prediastolic" bruit was audible, apparently connected in the first case with the aortic, and in the second case with the pulmonic valve. For permission to make use of the first case I am indebted to the kindness of Dr. Stone, under whose care the patient came; the second case was admitted into Dr. Peacock's ward during his temporary absence, and remained for a short time only in the hospital. The first case was under observation for a considerable period.

*CASE 5.—Morbus cordis; heart enlarged; a diastolic and a variable systolic and "presystolic" bruit heard at the apex; a short "prediastolic" bruit at the base; œdema of the lower parts of the body; great ascites.*—Alicia C., æt. 43, married, was admitted into St. Thomas's Hospital under the care of Dr. Stone, on March 28th, 1876.

Twenty years before she had had an attack of rheumatic fever, after her second confinement; she was unable to walk for eight months. Since her last confinement, only eighteen months ago, she has found her breath getting short, and three weeks after that event she first noticed some swelling about her legs and abdomen, which gradually increased. She obtained temporary relief under treatment as an in-patient at St. George's Hospital, and subsequently she improved very rapidly while under Dr. Stone's care in St. Thomas's Hospital between September 29th and October 30th, 1875. I am unable to obtain the notes taken of her condition at this time, but according to my recollection, there was a difference of opinion as to whether the bruit then heard at the apex was systolic or "presystolic" in character. It had been put down as "presystolic" at St. George's Hospital; and I have little doubt that, when observed by us, it presented sometimes one character and sometimes the other. The dropsy and ascites and general distress had been increasing again during the nine weeks previous to her readmission in March of the following year. At this time the abdomen was greatly distended, measuring forty inches in circumference at the umbilicus. Some rhonchus was heard with the respiration all over the chest, but there was no dulness to percussion over the

lungs; no enlargement of the liver could be detected. The urine was scanty, of a sp. gr. 1025, with a deposit of lithates, but containing no albumen. The heart was enlarged, the apex beating in the axilla. On March 27th Dr. Stone noted at the apex a diastolic murmur, with an occasional systolic murmur, and a systolic murmur audible inside the nipple. On March 28th I made the note:—At the apex (in the axilla) no distinct bruit; between the nipple and sternum a systolic and a "prediastolic" bruit are heard, with a "presystolic" thrill. At the base the "prediastolic" bruit is harsh, running up to a sharply accentuated second sound.

On April 1st I made a somewhat fuller note:—At the apex, beating in the axilla, a sharp bruit is heard running up to a loudly accentuated first sound; here also a distinct thrill is felt with the impulse, and terminating abruptly with it. To the right of the nipple the first sound appears merely prolonged, but here a rough sound is heard preceding and running up to the second sound, simulating closely that audible before the first sound at the apex. At the base this "prediastolic" bruit alone is audible; it is very distinct over junction of third left costal cartilage with the sternum, where the second sound is very sharp and accentuated; pulse 78, weak. This bruit running up to the second sound was constantly listened to by me, and its character and relation to the other cardiac bruit frequently noted.

On April 10th I made a note:—At the apex is a "presystolic" bruit with a thrill, the first sound is loud and sharp, the "prediastolic" bruit is faintly audible. At the end of the sternum a systolic and "prediastolic" bruit are audible, the second sound being sharp. At the base a systolic and the "prediastolic" bruits are heard; the former is also conducted along the aorta.

On April 12th the prediastolic bruit is noted as being distinct over the region of the pulmonary artery, and a slight thrill was felt there. The bruit at the apex was systolic in character.

April 21st.—Paracentesis abdominis was performed to relieve the increasing distension, fifteen pints and a half being withdrawn.

26th.—The "prediastolic" bruit was very distinct at the

base, less so over the aorta and down the sternum; it was audible at the apex, and at this point a systolic bruit of variable intensity was heard, sometimes not distinguishable; no distinct thrill was felt.

May 6th.—The position of the apex was noted as one inch and a half outside the nipple, and on a level with it, but the situation of chief impulse not well defined. In the axilla a well-marked and characteristic "presystolic" bruit was heard, running up to the first sound; the second sound seemed to follow quickly after the first; it was reduplicated, and was marked by a soft prolongation; the first sound was sharp and accentuated. Higher up, and loudest at the base, was heard the "prediastolic" bruit, running up to the second sound. Here there was also audible a slight systolic bruit; there was no systolic bruit at the apex. Finally, it was especially noted that the heart's action was deliberate, and the "grand silence" long.

10th.—No distinct bruit was audible with the first sound, and no thrill felt. The "prediastolic" bruit was audible at the base as before; very strongly resembling a "presystolic" bruit at the apex.

17th.—There was a short "presystolic" bruit at the apex, the "prediastolic" bruit being indistinctly heard at that point. The "presystolic" bruit was very distinct at the sternum, where also the first sound seemed prolonged (? a slight systolic bruit). The "prediastolic" bruit was loudest at the base.

20th.—A "presystolic" bruit heard, limited to the region of the apex; inside and below the nipple a slight systolic bruit was heard over a limited area. At the base, as well as the "prediastolic" bruit, a slight creaking sound was heard during the systole: a slight thrill was again felt at the apex.

24th.—Quite at the apex (in the axilla) a "presystolic" bruit was heard, short, and running up to the first sound, and a diastolic bruit, also short, following the second sound. With the "prediastolic" bruit at the base a slight systolic bruit was heard; the former was loud over the sternum, and between this and the nipple.

This patient left the hospital on June 15th, much improved. The somewhat detailed account given of this case will serve

to show how constant the "prediastolic" bruit was in comparison with the variability of the bruit associated with the mitral valve, on account of which the case presented no little complexity in its auscultatory phenomena. But however the other phenomena changed, the short "prediastolic" bruit maintained its peculiar and distinctive character throughout. The only feature of this case, which seems capable of throwing any doubt upon the question of the causation of this bruit, is the indication of the existence of some old fibrous adhesions about the base of the heart and the root of the aorta. But opposed to the bruit being regarded as exocardial rather than endocardial in origin, is not only the fact of its exact relationship to the second sound, but the unequivocal fact of its being transmitted in the direction of an ordinary aortic regurgitant bruit.

*CASE 6.—Systolic and "prediastolic" bruits audible over the pulmonary valve.*—Sarah B., æt. 20, single, and in service, was admitted into Alice ward on June 5th, 1876, under the care of Dr. Peacock.

Her father is a shepherd, he is alive and healthy; her mother died at an age, and from causes, unknown to the patient. She has three sisters and one brother all strong and healthy, and all taller and bigger than herself; she is youngest but one. She stated that her mother never suffered from rheumatism; there has been no rheumatism or gout in her family. She has herself always been strong and enjoyed good health: she has had no previous illness that she can remember. Her breath has, however, always been rather short on exertion, as in running.

When admitted, an attack of acute pleuro-pneumonia on the left side, which had come on quite suddenly four days previously, was just past its crisis. She had been taken all at once with shivering and vomiting, and with a severe catching pain in the left side. She had been obliged to take to her bed at once, and had kept it up to the morning of her admission, suffering in the meantime from much fever, some looseness of the bowels, and cough, but without expectoration. On the day of her applying for admission she was feeling much better; and the temperature, which was high in the

morning, fell in the course of the day, and reached the normal point in the evening, and near this point it subsequently continued, the patient making a rapid convalescence.

On examining the chest it was found that disease of the pulmonary valve existed, as an accidental complication of the case, in addition to the consolidation of the base of the left lung, with effusion of lymph on its surface.

On a subsequent occasion, by a more particular examination of the cardiac region, the following conditions were made out:—The impulse of the heart could be felt to a distance of half an inch outside the mammary line, and about an inch below the level of the nipple, under the fifth rib. The cardiac dulness extended upward to the upper border of the third costal cartilage, it was limited to the right by the left border of the sternum. There was a strong pulsation in the fourth interspace, between the left nipple and the sternum, which seemed to indicate some hypertrophy of the right ventricle. A marked thrill was felt in the second left interspace, occupying the whole of the systolic period, and terminating sharply with the closure of the sigmoid valves; no thrill could be felt elsewhere. On auscultation a very harsh and loud systolic bruit was heard at the junction of the third left costal cartilage with the sternum; this bruit, after diminishing in intensity, became again intensified, and ran up to and was brought abruptly to an end with the second sound, which seemed abnormally accentuated. Both bruits were distinctly transmitted to the left in the course of the pulmonary artery. A systolic bruit was heard also down the sternum, and was distinctly audible at the lower end of that bone, but here it presented nothing of the harshness of the basic bruit, and was probably attributable to regurgitation through the tricuspid valve, evidence of which was shown in a distinct pulsation in the veins of the neck. A slight systolic bruit was also heard at the apex of the heart.

This compound systolic and "prediastolic" bruit over the pulmonary valve and artery, was frequently observed during the patient's residence in the hospital, throughout which period its character remained constantly the same. The patient left the hospital on June 23rd.

In both these cases the impression conveyed was most distinct, that from some cause a defective closure of the semi-lunar valves occurred. The rarity of the bruit would seem to present the chief difficulty in the way of accepting this view of the case. The question naturally suggests itself, whether there is anything to account for the fact, that such an indication of partial failure of the semilunar valves is so seldom observed, while the bruit indicative of complete insufficiency of the valve is continually met with, and while again the very common occurrence of the "presystolic" bruit would be regarded, from the same point of view, as indicating the no less frequent occurrence of a corresponding condition of partial defect of the mitral valve. It might appear inconsistent to associate a phenomenon of such rare occurrence with a physical condition, of a kind which must presumably be very commonly present in a greater or less degree. The rarity of a distinct "prediastolic" bruit may, however, be taken on the other hand as indicating the rarity of the conditions, under which a slightly defective state of the semilunar valve is productive of an appreciable murmur. That the impeded closure of the semilunar curtains should necessarily be attended with less regurgitation, than would be permitted by a corresponding impediment in the case of the mitral valve, is in accordance with the essential difference in the forces, under the influence of which the closure of the two valves takes place. The force of the recoil of the blood driven into the aorta and great vessels, is truly impulsive in its nature. The blood issuing in a rapid and forcible stream from the aortic orifice of the ventricle, leaves the first part of the aorta in a state of comparative relaxation, being driven forwards to distend this vessel, and the great arterial trunks, further on; the arrest of its forward movement resulting in a conversion of its initial momentum into a distending force. At the conclusion of the ventricular systole, the resilience of the aorta and great vessels, no longer opposed by a continued afflux from behind, will prevail to drive a part of the blood wave backwards against the aortic valve, upon which it will come with some degree of acquired momentum, closing the curtains instantaneously by the force of its sudden impact. A degree of valvular thickening and rigidity, incapable of producing

more than a modification of the sound of the impact of the semilunar curtains closed by such a force, might well suffice, in the case of the mitral valve, to produce a distinctly recognisable "presystolic" regurgitant bruit, in the presence of the gradually, if rapidly, exerted ventricular force; and the more so, if the heart's action should be slower than normal. But, as before observed, if the "presystolic" bruit is of frequent occurrence, a "roughening" of the second sound, presenting the rudiment of a "prediastolic" bruit, and indicating a slight resistance to its closure, is, as I believe, not uncommonly to be detected.

What condition may have been present in the above cases, to cause a more distinct "prediastolic" bruit to be produced, it may be difficult to determine. It is possible that this may have been due to some exceptionally unfavorable position of one or more of the curtains; since if the semilunar curtains, while becoming rigid, are drawn inwards by adhesion at their cusps, they will be brought into a position in which they will be more directly subjected to the force of the blood, a result which might fully compensate for the effect of this rigidity, and permit the closure of the valve without any bruit being produced. It is to be observed, moreover, that such a slight defect in the closure of the semilunar valve is a matter of no inconvenience to the patient, and of little or no importance to him; and if, at the same time, it is for the most part attended by such slight indications, it is likely to be passed over without comment.<sup>1</sup>

Adopting the view of Dr. Walshe as the only intelligible explanation of the "prediastolic" bruit, the two cases above recorded afford inferential evidence of the truth of Dr. Barclay's view of the causation of the "presystolic" bruit, of no little force.

For if a condition of the semilunar valves may exist capable of producing a distinct bruit of limited regurgitation, checked by a later impact of the curtains, much more frequently must such an impediment to the closure of the mitral valve, so often

<sup>1</sup> In the above cases I should myself, probably, not have taken such especial notice of the bruits described, had it not happened that my attention was, at the time, particularly directed to the observation of cardiac murmurs of this peculiar character.

presented in a rigid condition of the curtains, be attended by a similarly arrested regurgitant bruit, the physical conditions for the production of a bruit under such circumstances being so far more favorable in the case of the latter, than in that of the former valve.

Reviewing the different facts and observations, which have now been considered in relation to the physical cause of the "presystolic" bruit, they seem to me to afford very strong evidence that this is really due to mitral regurgitation, and that the auricular systolic hypothesis is scarcely tenable. The ground on which this conclusion is based may be thus briefly summarised :

1. The character of the bruit heard in certain cases of mitral stenosis, when regarded in connection with the features of the cardiographic tracings taken from the patient, is seen to be directly incompatible with certain assumptions, as to modifications in the character and mutual relations of the auricular and ventricular contractions, which appear to be necessarily implied in the auricular systolic hypothesis obtaining in these, or in other cases.

2. A consideration of the mechanism of the closure of the mitral valve shows that, where the mitral curtains are rigid, as in cases of mitral stenosis, or where they are drawn out of position, or where they may be, more or less on both accounts, presented under less favorable conditions for their immediate closure, by the pressure of the blood, when the ventricle contracts, but where the curtains are still capable of being effectually brought into contact by this force,—as is indicated during life in cases of "presystolic" bruit, by the sharpness of the first sound, and confirmed in some cases by testing the competence of the valve after death,—conditions are presented which must necessarily tend to produce, in greater or less degree, a bruit of this precise character by limited regurgitation through that orifice ; and it is further clear that the absence of any indication of the occurrence of such regurgitation under these conditions, could only be explained by a simultaneous augmentation of the rapidity of the ventricular systole, of which there is no constant evidence, while a slower contraction of this cavity, corresponding with the deliberate character of the heart's action often con-

currently observed, must tend to develop with a proportionate degree of prominence, a regurgitant "presystolic" bruit so produced.

8. The occurrence of the "presystolic" bruit independently of mitral stenosis is quite in accordance with this view of its causation, but it is incapable of explanation as the effect of the auricular systole. As an indication of incomplete failure of the mitral valve, its occurrence fills up an apparent hiatus in the consecutive cardiac phenomena.

4. The occurrence of a short "prediastolic" bruit of an analogous character, attributable only to partial failure of the aortic or pulmonic valves, would scarcely accord with the absence of a corresponding indication of similar failure of the mitral valve, under circumstances far more favorable to its prominent development.

#### *IV. Facts which have been regarded as opposed to the views advanced by Dr. Barclay.*

I shall now finally make some reference to those objections brought forward in opposition to Dr. Barclay's view, which seem to me to have the most direct and important bearing on the question. Some of these have been already incidentally alluded to. The sharpness of the upstroke of cardiac tracings from cases of mitral stenosis has been regarded as evidence of a rapidly contracting ventricle, but it was pointed out that this is quite consistent with the "presystolic" commencement of a sluggish ventricular systole; and this sluggishness of the contraction of the ventricle will account for an absence of any notable shortening of the systolic portion of the tracing, such as might have been expected to occur when the impulse and first sound are postponed to a later period of the systole. Reference has also been made to another fact which has been regarded as opposed to this view, namely, the loudness and comparative softness of the two portions of the continuous "presystolic" and systolic bruit, which is not unfrequently heard in cases of mitral stenosis. It was pointed out that this change in the character of a continuous regurgitant bruit is attributable to the effect of contact of the mitral curtains

oy their edges, at the moment of the "systole." It is less easy to understand the explanation of this fact given on the auricular-systolic hypothesis, that the loudness of the auricular direct bruit compared with the ventricular regurgitant bruit, notwithstanding the feeble force exerted, is due to the fact that in the former case, but not in the latter, "the muscular force, as developed in the blood currents, converges on the orifice through which the blood is forced."<sup>1</sup>

Another fact which has been regarded as opposed to Dr. Barclay's views is that, in cases of "presystolic" bruit, the impulse, the first sound, and the carotid pulse, maintain their normally close relationship to each other. It is argued that "a ventricular contraction, capable of educing a valvular click, and of presystolic rhythm, should necessarily be accompanied by a presystolic pulsation of the aorta and its primary branches; hence the carotid pulse should precede the first sound."<sup>2</sup>

In regard to this it may be pointed out that it has been demonstrated by Marey's observations, that the aortic valve is not opened until the intra-ventricular pressure has nearly reached its maximum point. Probably from the very commencement of the ventricular contraction, there is a continuous rise in the intra-ventricular pressure. It is manifest, however, that this cannot reach any important degree, until the mitral valve has been closed (efficiently or inefficiently); not until after this has occurred, will the ventricle effectively grasp the blood within it, in the process of which its cavity becomes rounded, while its walls become tensely stretched over their contents; and a still further contraction is required for a diminution of its capacity and the expulsion of the blood. The precise order of events must therefore be, closure of the mitral valve, the occurrence of ventricular tension, with the impulse or pressure of the ventricle against the chest wall, and lastly the carotid pulse. Owing to the rapidity with which, under normal conditions, the ventricular contraction reaches its full power, these phenomena succeed each other in such rapid succession, as to be indistinguishable in the time of their occurrence. But if the ventricular contraction were sufficiently slow, these

<sup>1</sup> Dr. Balfour, 'Lancet,' May, 1872, p. 715.

<sup>2</sup> Dr. Hayden, 'Diseases of Heart and Aorta,' p. 898.

phenomena would follow each other at appreciable intervals, the valvular impact, and ventricular tension, preceding the pulse: a rigid state of the mitral curtains, on the other hand, would under these circumstances, by delaying the valvular impact until a later, and therefore more active, period of the ventricular systole, tend to bring about a re-approximation in rhythm between the first sound and the impulse and carotid pulse. It does not, however, seem easy to understand how such a delayed closure of the valve could lead to a reversion of this order of events. With the closure of the mitral valve, which gives its *point d'appui*, the real work of the ventricle begins.

Two important characteristic peculiarities of the "presystolic" bruit, both of which appear in some respects opposed to Dr. Barclay's view, still remain to be considered. These are, 1, the limited localisation of the bruit to the immediate neighbourhood of the heart's apex; and 2, its remarkable instability.

1. The fact of the bruit being so loudly audible at the apex is immaterial to the question of its causation, since the ordinary bruit of mitral regurgitation may be heard at the same point with great intensity and harshness.

The real objection to regarding the bruit as regurgitant lies in the fact that, although heard with such intensity at the apex, it is very rarely audible in the back at all. In considering the validity of this objection, it will be necessary to enter into some discussion of the broader, and much-debated question of the apparent discrepancy, which exists between the auscultatory indications of cardiac valvular lesions, and the results of experimental observations made with a view of elucidating them. The results of Sir D. Corrigan's experiments are clinically illustrated by the fact that the systolic bruit of aortic or pulmonic obstruction may be scarcely audible over the cardiac area behind the semilunar valves, even if very loudly heard over the aorta or pulmonary artery; and the fact that the bruit of mitral or tricuspid obstruction, though loud at the apex, are similarly inaudible in a backward direction, is also in agreement with them. On the other hand these experiments do not in any degree explain the fact that the bruit of aortic insufficiency may be sometimes but

little less loud and harsh over the aorta than down the sternum, or that the mitral regurgitant bruit is heard so loudly and harshly at the apex.

It has been suggested that the loudness of the mitral regurgitant bruit at the apex might be due to the sound being conducted to the point along the walls of the ventricle. It will, however, I think be found, that the apparent inconsistency between the last-mentioned clinical facts and the experimental result, is sufficiently explained by a consideration of the element of valvular vibration, present in the generation of the cardiac murmurs, but absent in those experimentally produced, the introduction of which constitutes the essential difference in the physical conditions presented in the two cases.

While the experiments of Sir D. Corrigan clearly prove that the fluid vibrations, developed on that side of the valves towards which the blood is driven, are very greatly interfered with in their transmission backwards—whether by the mere effect of the narrowness of the opening, or possibly also by a loss of conducting power of fluid in a state of extreme molecular agitation, such as must attend its sudden passage from a state of constraint to one of comparative freedom—it is no less clear that the valvular vibrations simultaneously produced, must be communicated with equal force to the blood immediately in contact with the curtains on either side; and that, if the conditions for their transmission from each surface of the valves were equally favorable, they would be conducted with equal intensity in both directions. And the question to be considered amounts to this, whether the semilunar and mitral valves present features in their construction, which suffice to explain the facts, that the vibrations of the former may be distinctly audible only in the direction of the aorta or pulmonary artery, as in certain cases of obstruction; and that, as in the case of the mitral obstructive bruit, the vibration of the mitral valve may be heard only at the apex of the ventricle.

If we consider the position of the mitral curtains, sloping down towards each other from the margin of the auriculo-ventricular aperture, in the upper part of the ventricular cavity, it might be anticipated that the vibrations communi-

cated from the curtains to the confined wedge-like mass of fluid situated between them on their auricular surfaces, would tend to interfere with each other in greater or less degree, being propagated in directions more or less opposed to each other, and that the valvular element of any bruit transmitted through the auricle would be, on this account, comparatively insignificant. On the other hand the free space of the ventricular cavity, in which the curtains stand prominently forward, presents a condition especially favorable to the effective transmission of the valvular vibrations in this direction. The position of the aortic valves must tend in a similar manner to favour the transmission of the vibrations of the semilunar curtains in the course of the aorta, while correspondingly unfavorable to their transmission in the opposite direction. In any case, however, the degree in which the transmission of the vibration, whether valvular or of a purely fluid character, is favoured, or otherwise, by the physical conditions, must necessarily vary with the exact nature of the valvular lesion present. From the results of Sir D. Corrigan's experiments, then, and from the arrangement of the valvular curtains, it would be concluded that, 1, in the case of regurgitation through either the mitral or aortic orifices, the bruit heard in the direction of the returning stream will be mainly due to fluid vibration, while that heard in front of the valve will be mainly due to the transmission of valvular vibrations; and, 2, that bruits due to obstruction at either orifice will be more loudly audible in front of the obstruction on both accounts.

This does not explain the remarkable difference between the intensity with which an aortic regurgitant bruit may be heard down the sternum, and the indistinctness, and in many cases the inaudibility, near the spine, of a mitral regurgitant bruit heard loudly at the apex. This great diminution in the sounds is greater than the separation of the left auricle from the spinal column can well account for, and it would seem to indicate that the fluid vibrations themselves must be more intense in the case of aortic than in that of mitral regurgitation. This inference is borne out by the presence, in very unequal degree in the two cases, of a most essential requirement for the production of fluid vibrations, *i.e.* the freedom of the space into which the fluid is projected; a condition equally

important, as regards the loudness of the sound produced, with either the degree of constriction of the aperture, or the magnitude of the propelling force. In the case of aortic regurgitation, if the ventricles be acting efficiently, still capable of expelling its contents completely, and if no congestion of the pulmonary veins is present, the integrity of the mitral valves still protecting the lungs from pressure, not only will there be no impediment to the blood returning through the aortic valve, but the elastic resilience of the ventricular walls may exert an exhaustive influence, still further increasing the difference between the pressure on the two sides of the valve; and hence the fluid bruit produced will, under such circumstances, be very loud in proportion to the other condition present. With failure of the ventricle, however, and with increasing engorgement of the left auricle and pulmonary veins, this condition will be less and less favorably presented, and quite independently of any aggravation of the valvular lesion, the bruit must thus become less and less loudly developed. In the case of mitral obstruction, the aortic valves remaining competent, the resilience of the ventricular wall will be brought to bear with its full effect on the blood flowing in from the auricle, and in this case moreover a further condition favorable to the development of fluid vibrations, is presented in the more or less conical projection into the ventricular cavity formed by the adherent mitral curtains. A stream of blood issuing from a constricted mitral orifice, at the apex of such a cone, into the empty ventricle, tending to expand by the resilience of its walls, will manifestly be under conditions the most favorable for the production of fluid vibrations, and hence its loudness in proportion to the comparative feebleness of the force producing it.

But in the case of mitral regurgitation the condition so favorable in the above instances is entirely wanting. The left auricle, never empty, and incapable of exerting any expansive force, is the very organ upon which the strain on the circulatory system, resulting from failure of mitral sufficiency, immediately falls; and with an increasing degree of permanent auricular repletion, the condition becomes less and less favorable to the production of fluid vibrations in the stream of blood regurgitating into it; and in this manner the comparative

feebleness or absence of this bruit in the back may be in great measure accounted for.

But the conditions affecting the production of fluid vibrations are in some respects still more unfavorable in the cases of "presystolic" than of systolic or "postsystolic" regurgitation through the mitral aperture.

The freedom of the "presystolic" regurgitation through the still open valve, compared with that occurring after the curtains have come into contact, through chinks between them, must affect the generation of fluid vibrations in two ways at least. For not only (1) will more vigorous fluid vibrations be produced in the issuing stream, in proportion to the narrowness of the exit, the pressure behind remaining the same, but (2) in respect of the ventricular contraction, in proportion to the freedom with which the blood can escape into the auricle, will the intra-ventricular pressure fail to rise in correspondence with the force exerted.

In the case of presystolic regurgitation through a contracted mitral orifice, however, there is, for the most part, another condition especially unfavorable to the development of fluid vibrations, presented in the state of mitral curtains themselves, and in their position at the time that this presystolic regurgitation occurs.

The arrangement of the curtains both of the mitral and of the sigmoid valves, must be in some degree less favorable to the development of vibrations in the stream of regurgitation, than in that flowing directly forwards through either opening, since they present to the former, after it has passed through the chink between their edges, a gradually widening channel, if it be but for a short distance, while they cause the latter to issue from a projecting aperture. Although the loudness with which the bruit of aortic regurgitation is heard down the sternum would seem to indicate that here, at all events, the effect of the position of the valves is practically unimportant, or obviated, it may be, by eversion of the edges of the curtains (and it is probably the same in the case of regurgitation through simple incompetence of the mitral valve), this condition will be presented in a much more distinctly unfavorable manner when the mitral curtains have become intimately united with each other in the shape of a more

or less completely formed, funnel-like or conical passage ; and when, moreover, instead of being thrust inwards with their free margins in apposition, and bulged backwards by the pressure upon them, as during the occurrence of systolic ("postsystolic") regurgitation, the mitral curtains stand apart so as to make a longer and more uniformly expanding channel. Such a state and position of the valvular curtains will be unfavorable to the development of fluid vibrations in the case of regurgitation, in a manner and to a degree corresponding with its opposite influence in favouring the development of vibrations in the "direct mitral" blood stream flowing in the opposite direction.

On the other hand an open state of the mitral valve, with the edge of the curtains directly meeting the blood stream forced past them, is a condition favorable to the generation of vibrations in these curtains themselves ; while at the same time an open, as compared with a closed state of the valve, will be as much less favorable to the conduction backward of such valvular vibrations as to the generation of fluid vibrations in the back-flowing stream, and for the same reason, that their surfaces are more directly opposed to each other under the former than under the latter conditions.

It thus seems to me to be no matter of surprise that a bruit so curtailed in its duration, and produced under conditions in several respects physically unfavorable to its transmission in a backward direction, should be distinctly audible in the dorsal region under very exceptional circumstances only ; and very rarely in comparison with the frequency with which the ordinary mitral systolic murmur is heard in the same position.

A comparison of more accurate observations of the condition of the valvular curtains in different cases, and of the clinical phenomena associated with them, will afford a fuller elucidation of this difficult subject than that here attempted in part. It seems to me, however, that such consideration as the above, though embracing some only of the physical condition affecting the question, yet suffice to show that the fact, that the "presystolic" bruit is audible in the neighbourhood of the heart's apex alone, cannot be regarded as incompatible with its being due to mitral regurgitation, and that this fact in no degree invalidates the inference deducible from physiological

and clinical observation on points less complex in their nature and better understood.

2. A recognition of the essentially "*valvular*" character of the "presystolic" bruit renders certain phenomena observed in connection with its variability, and more especially its absence under conditions apparently most favorable to its development, facts which would otherwise appear scarcely compatible with Dr. Barclay's view, capable of explanation in a manner fully according with it.

The degree of variability of this bruit is remarkable and characteristic. It is found to be interchanged with a systolic bruit, as in some of the cases above mentioned, or to disappear entirely from time to time, under varying conditions of the patient's general health, or from day to day from causes less obvious. When thus absent it may often again become distinctly audible on the excitation of the heart's action by slight exertion on the part of the patient. In other cases, though distinctly heard when the heart is acting quietly and with deliberation, it may cease to be distinguishable if the cardiac action becomes excited.

It might not seem difficult to account for these phenomena by variations in the effectiveness of the auricular contractions under the varying conditions of the cardiac functions. The disappearance and reappearance of the bruit, with a diminished or increased vigour of the heart's action, might be referred to a directly corresponding variation of the vigour of the auricular systole; and the disappearance of the bruit as a result of cardiac excitement might equally well be attributed to a weakening of its contraction from the paralysing effect of over-distension.

It would appear, however, more in conformity with the facts, to refer such marked variations in the physical phenomena, to variation in the force of the more powerful ventricular contractions—a force having a range of action greater than that of the comparatively feeble auricular systole, in proportion to its far greater power. It is difficult to suppose that the auricle, at one time contracting with a force insufficient to produce any audible vibrations in the auriculo-ventricular blood-stream, should, by the excitement attendant upon the patient's merely walking up and down a hospital ward, be

stimulated to such increased activity as to generate a harsh and loud "presystolic" bruit.

But if the degree of the variability of the "presystolic" bruit corresponds with the wider variations of the force of the ventricular contractions, the phenomena themselves appear to be capable of as clear interpretation by reference to this force as by reference to that of the auricular systole.

An increased activity of the ventricular contraction is, as before pointed out, a condition which tends directly to counteract the effect of rigidity of the mitral curtains in producing a "presystolic" regurgitant bruit; this may abridge or even render indistinguishable a well-marked bruit of this character, distinctly audible when the heart's action is slower. To account for the disappearance of such a bruit under the exactly opposite condition of *enfeeblement* of the ventricular contraction, it has to be remembered that, in order to produce vibrations in thickened mitral curtains, of sufficient rapidity to elicit audible sounds, a certain relation must necessarily exist between the greater or less degree of immobility of these last, and the force of the ventricular contraction brought to bear upon them; so soon as this force falls below the required point, the bruit, if dependent upon the vibration of the valvular curtains, as was inferred above on physical grounds, will cease to be heard. In some instances it appears that a transient "presystolic" bruit may be thus developed on excitation of the cardiac action, owing to a consequent dilatation of the ventricle, a condition shown to be sufficient to account for the effect observed.

From the absence of such a relation between the thickening of the mitral curtains and the power of the ventricles, resulted, in all probability, the phenomena presented in the cases from which the tracings *c* and *d* (p. 187) were taken by Dr. Galabin. Unless the explanation afforded by the recognition of the essentially "valvular" character of the regurgitant "presystolic" bruit is taken into consideration, it might well be thought that the absence of any bruit in the true "presystolic" period, in cases in which the presence of a prolonged and more or less harsh diastolic mitral murmur gave certain evidence of the existence of a high degree of mitral stenosis, would be scarcely compatible with Dr. Barclay's

views. If the pressure of the blood accumulated in the auricle sufficed to produce the loud diastolic bruit heard immediately following the second sound, how could it have happened, on Dr. Barclay's hypothesis, that the far more powerful ventricular contraction should have failed to produce a "presystolic" regurgitant murmur of proportionately increased intensity? Attributing this bruit to the transmission of the valvular vibrations alone, the fact that the diastolic mitral bruit was followed in the former instance by a clear first sound, might be taken to indicate that in this case the curtains if thickened were capable of being closed by a force insufficient to throw them into vibration sufficiently vigorous to produce a bruit, while the systolic bruit which followed the diastolic bruit in the second case would indicate that this was effected by the full force of the ventricular systole, though not at its commencement. It may have been, on the other hand, that in these exceptional cases, the mitral curtains, though much adherent, may yet have retained considerable pliability, or may have been drawn into a position in which the ventricular pressure would bear upon them with greater advantage in bringing them into contact; and that on either account, or on both conjointly, the valve was still capable of being closed without appreciable regurgitation.

On the auriculo-systolic hypothesis it is not easy to understand why no properly "presystolic" auricular contraction occurred in these cases, although an earlier, but presumably, on this hypothesis, a subsidiary contraction was distinctly indicated, both in the character of the bruit and in the cardiographic tracing.

None of the facts here considered can I admit as presenting any serious obstacle to the acceptation of Dr. Barclay's views, which seem to be so fully supported on other grounds. Whatever weight may be attached to them, the obscurity which surrounds the questions which they involve, is such as to sink their importance, when contrasted with the deductions from the distinct results of auscultatory and cardiographic observations, before adduced as controverting the views laid down by M. Fauvel and Professor Gairdner on this subject.

Adopting Dr. Barclay's view as to the causation of the "presystolic" bruit, and a similar view of the analogous

"prediastolic" bruit, the enumeration of uncomplicated cardiac bruits given by Professor Gaiderner in his 'Clinical Medicine' might be indicated in an extended form as follows:—

a. Murmurs audible at the apex, and produced at the mitral or tricuspid orifice.

1. "Presystolic," <i>running up to the first sound</i> . . . .	=	Partial incompetence (associated with stenosis in general).
2. Systolic, <i>running off from first sound</i> . . . .	=	Complete incompetence.
3. Diastolic, <i>running off from second sound, or quickly following it.</i> (Commonly having an auricular-systolic intensification, and very rarely uncomplicated with a "presystolic" bruit.) . . . .	=	Stenosis or roughening of the valve.

b. Murmurs audible at the base chiefly, and produced at the aortic or pulmonic orifice.

1. "Prediastolic," <i>running up to second sound</i> . . . .	=	Partial incompetence.
2. Diastolic, <i>running off from second sound</i> . . . .	=	Complete incompetence.
3. Systolic, <i>running off from first sound</i> . . . .	=	Stenosis, or roughening of the valve.

The list of murmurs thus arranged presents a complete and symmetrical appearance. The introduction of the "presystolic" and "prediastolic" bruits, with the above interpretation, fills up what would otherwise appear to be an unaccountable gap in its consecutiveness.

## ANATOMICAL VARIATIONS.

(II.)

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THE following are a few of the rarer varieties noticed in the dissecting room of this Hospital in the past session 1876-77, during which time thirty-six bodies have been dissected.

*Large bony growth projecting from the middle of the front of the humerus, and attached by ligament to the front of the coronoid process of the ulna.*

This variation seems worthy of record, perhaps more from its surgical interest than for any other reason. It occurred in the right arm of an exceedingly muscular male subject, æt. 71, whose occupation was unknown. It may, however, be conjectured he was a labourer, accustomed to carry heavy weights on his back, from the increased thickness and coarseness of the skin and subcutaneous tissue over the lower cervical and upper dorsal regions.

On examining the right upper extremity, the first thing noticed was that the forearm could not be completely flexed on the arm, but stopped short about midway between complete and rectangular flexion, on account of a large bony growth

projecting from the middle of the front of the arm, and in flexion pressing on the front of the soft tissues of the upper part of the forearm. It was also observed that the elbow could not be completely extended, on account of a band, easily felt underneath the skin, extending from the apex of this growth to the bottom of the bend of the elbow.

On further dissection the bony growth appeared completely concealed in the substance of the brachialis anticus muscle, and consisted of a large triangular process, four and a half inches long, and one eighth of an inch thick in most of its extent. Its base was continuous with two and a half inches of the anterior border of the shaft of the humerus, immediately below the deltoid eminence, and its apex pointed downwards and gave attachment to a ligament, which was fixed below to the tubercle and lower part of the front of the coronoid process of the ulna. The process was flattened from side to side so as to present an external and internal surface, and an anterior and posterior free border. The external surface was about three fourths of an inch broad at its broadest part, more or less smooth, a slight groove separating it from the external surface of the shaft, and at the junction of the upper and middle one third presented a small foramen communicating with the opposite surface. This surface gave attachment to the brachialis anticus in its whole extent. The internal surface was rougher and more hollowed than the external, and presented at the upper part the foramen already mentioned, and at the lower part a depression leading into a foramen for the passage of a nutrient artery. It also gave attachment to the fibres of the brachialis anticus in its whole extent. The anterior border, four and a half inches long, was more or less convex, and presented two lips with an intervening space gradually tapering towards the apex. The inner lip was much the more prominent and overhung the hollowed inner surface. This border, also, afforded attachment to the fibres of the brachialis anticus.

The posterior border of the freely overhanging process was one and a half inches long, thin and sharp and generally concave, and had attached to it a delicate connective-tissue septum, which blended with the ligament stretching from the apex of the process, and was attached to the anterior

border of the humerus from the base of the process to the anterior ligament of the elbow-joint, with which it became continuous, and thus *completely divided the brachialis anticus into two halves.*

Stretching from the apex of the process to the tubercle of the ulna and lower part of the anterior surface of the coronoid process, there was a strong ligamentous band (represented in the sketch by two parallel dotted lines) three and a half inches long and about half an inch thick, into the lower two thirds of the sides of which fibres of the brachialis anticus were inserted and from the upper one third a number of them arose. In extending the elbow-joint this band became so tight as to prevent its complete extension.

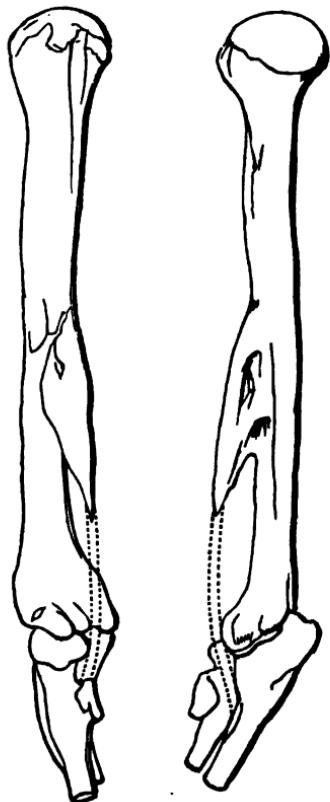
There were slight apparent evidences of rheumatic arthritis from the greater size and overlapping character of the tuberosity of the radius, and from the presence of a small nodule of bone about the size of a pea, connected with the inner surface of the capsule of the elbow-joint on its outer aspect. There was, likewise, increased roughness round the head of the humerus, and the long tendon of the biceps was partly adherent to the inner surface of the capsule of the shoulder-joint and partly arose from the upper extremity of the bicipital groove and adjoining surface of the lesser tuberosity.

The abnormality in this case must we think be looked upon as more pathological than physiological. It does not appear to find a homologue as do so many of these irregularities in any constant process of the kind in the lower animals, but must rather be considered a true exostosis developed in the substance of the brachialis anticus and along the course of the connective tissue between parts of the muscle.

It is perhaps hardly a matter of surprise that such a development should occur by preference between the two halves of the muscle. Some indication of such a division naturally exists in the upper part, where the deltoid passes down between the two halves, and in the agouti (*dasyprocta*) the muscle is permanently divided into two halves.

It presents a striking analogy to two preparations, in the museum of St. Thomas's Hospital (Nos. C 35, C 36), of a large exostosis arising from the front of the femur, and is like them also in the bony growth being connected with the middle of

the shaft of the bone. For the drawing of the preparation we are indebted to the kindness of Mr. Stewart.



Unusual bony process growing from the humerus and dividing the brachialis anticus.

#### *Double omohyoid muscle.*

On the left side of an average muscular female subject this variation was seen. It consisted of a muscle, fleshy in its entire extent, arising from the anterior surface of the hyoid bone internal to the attachment of the omohyoid, and inserted mainly into the posterior surface of the sterno-clavicular articulation, blending with the outer edge of the sterno-

hyoid, another portion passing with the posterior belly of the omo-hyoid. It was of the same thickness as the anterior belly of the omohyoid, and in its course presented a curve with the convexity directed backwards in the neck. For the first two inches of its length it ran parallel with, and was separated from the anterior belly of the omohyoid by a distinct fibrous septum, and at the point where the latter muscle crossed the carotid artery it divided into two unequal parts, the main part, consisting of about two thirds of the fibres, continued downwards, lying *on* the sheath of the carotid, to its insertion in the posterior sterno-clavicular ligament, the other smaller part passed backwards to blend with the posterior belly of the omohyoid, immediately beyond the intervening tendon.

This case differs slightly from those of double omohyoid hitherto recorded. Koster and Gruber mention two cases in which upper belly of the supernumerary muscle was split, and the inner piece joined the sternohyoid. Hallett and Sels each mention a case in which the lower belly of the extra muscle ran partly into the sternohyoid or sternothyroid respectively. But we do not find any instance on record in which the arrangement was the same as in the present case. This variety will be of surgical interest from the relation of the supernumerary muscle to the common carotid artery.

#### *Varieties in the stylohyoid muscle.*

In one case there was complete absence of the stylohyoid ligament on the left side. Its place was taken by a fusiform muscle, about one third of an inch thick at its widest part, and which, from its attachments might be called the *stylochondrohyoidens*. It arose by fleshy fibres from the apex of the styloid process, and passed downwards exactly in the course of the ligament to be inserted into the apex and sides of the lesser horn of the hyoid bone. The ligament was carefully looked for but nowhere to be seen. The muscle of the opposite side was normal.

It is interesting as showing that the epihyal bone of many animals, represented in man by ligament, occasionally more or

less ossified, may be represented in him by a muscle, and thus approach the arrangement found in some of the Edentata, viz. that "the hyoid bone is connected to the skull only by muscles" (Huxley, 'Anat. of Vert. Animals,' p. 336).

A similar muscle to that described here has been described by Drake ('Anthropologia,' Bk. iii, ch. 17), Weitbrecht ('Comment. Petropol.,' ix, p. 256), Blandin ('Nouveaux Éléments d'Anat.,' p. 874), and Gavarde ('Traité de Myologie Au.,' vii).

On the left side of a female body another curious variety was observed, and might be called the *stylo-hyo-pharyngeus*. The stylo-hyoid ligament was ossified about its centre, for half an inch of its length, and from this ossified part arose a distinct fleshy slip which passed downwards and backwards, slipping between the middle and inferior constrictors to be inserted into the side of the pharynx. In its course it passed over the stylo-pharyngeus muscle and glosso-pharyngeal nerve, and underneath the lingual artery.

#### *Extensor Pollicis et Indicis.*

This comparatively rare abnormality, which is so common among the Carnivora, was observed twice. It occurred in both forearms of one female subject and in the left one only of another. In both it had exactly the same anatomy. The muscle arose from the posterior surface of the ulna for the distance of two inches, between the attachments of the extensor secundi internodii pollicis and the extensor indicis. Its tendon having passed through a separate compartment in the annular ligament, split at the base of the first interosseous space into two equal parts, one passing outwards to blend with the tendon of the extensor secundi internodii, and the other to join the common expansion formed by the tendons of the extensor indicis and common extensor going to the forefinger. The muscle was fleshy from its origin as far as the upper border of the annular ligament, but below that point was tendinous, and had very much the same dimensions as the proper extensor indicis. Cases similar to the above have been described by Wood, Clason, and Macalister.

*Tibio Accessorius.*

This variation occurred in the left leg of a male subject, and consisted of a muscle about three quarters of an inch wide at its widest part, arising by fleshy fibres from about an inch of the middle of the posterior surface of the shaft of the tibia, internal to the origin of the flexor longus digitorum, and blending below with the inner head of the accessorius, just at the point where the external plantar vessel and nerve crossed over the latter muscle. In its course downwards it completely covered the posterior tibial vessels and nerve in the lower third of their extent, crossed over the tibialis posticus and flexor longus digitorum tendons, and passed through a special compartment in the internal annular ligament, between the flexor longus pollicis and longus digitorum. The muscle was fleshy from its origin to the upper border of the annular ligament, and below that point tendinous.

The variety is interesting from its relation to the posterior tibial vessels and nerve.

Macalister refers to this abnormality under the name of second flexor longus, but it appears to be extremely rare.



ON THE  
MUTUAL RELATIONS OF THE BIRTH-  
RATE AND DEATH-RATE.

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By J. S. BRISTOWE, M.D., F.R.C.P.

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It will probably be recollect that Dr. Richardson claimed as one of the benefits to be anticipated for those living in his visionary city "Hygeiopolis," that their annual death-rate should be reduced to '5 per cent. It will probably also be recollect that immediately after the reading of Dr. Richardson's celebrated paper an anonymous correspondent of the 'Times' asserted, with some show of argument, that a mortality of '5 per cent. implied an average duration of life of 200 years, and that, *a fortiori*, Dr. Richardson's anticipations were impossible of realisation and absurd.

There is no doubt that under one special condition (and that the condition which, on account of its simplicity, would probably present itself most readily for computation) the result arrived at by the 'Times' correspondent is correct. If an annual mortality of '5 per cent. be maintained in a population which neither diminishes nor increases, and in which the removals by death are exactly balanced by the additions by birth, it is certain that the average duration of life would be exactly 200 years. Thus, if in a population of 1000

persons five deaths and five births occur annually, and it be assumed for simplicity's sake that every individual of the population lives to the same (namely, the mean) age, it is obvious that exactly 200 years will elapse before the whole of the original 1000 will have died out, and that the turn of each annual litter to die will arrive exactly 200 years from the date of birth. But 200 years would still be the mean duration of life, even if the population were to die as they do now at different ages; only in this case while many would die below the mean age, many would exceed it, and some might easily attain to the years of Methuselah. The author of this calculation might have added that if a mortality of .5 per cent. were to be maintained in a population uniformly diminishing by that amount, the population would never die out; and that if it commenced with 1000 individuals the last whole man would live for somewhat over 1379 years, and would then perish only bit by bit in a diminishing geometrical series throughout all eternity. On the other hand, an annual death-rate of .5 per cent. with an annual birth-rate of 20,000,000 per cent. (a condition of things conceivable in the case of certain intestinal worms) would imply an average duration of life of little more than a year.

It is obvious that if with a uniform annual mortality of only .5 in the 100 the mean duration of life may vary, with variations of the birth-rate, from zero to infinity, even a low death-rate means *per se* neither healthiness nor unhealthiness, neither a long nor a short span of life, and that in order to determine its significance in these respects its relation to the birth-rate must be taken into account. It is because as an officer of health I have hitherto practically overlooked this important relation, and because, as I believe, the great majority of my colleagues have been equally neglectful—often, indeed, falsely boasting that while the birth-rates of our districts have been higher, and their death-rates only a little lower, than those of our less prolific neighbours, they were therefore in a twofold sense healthier than theirs—that I determined to investigate it. The results at which I have arrived form the more important part of the present paper.

The death-rate implies healthiness or unhealthiness only in so far as it implies long or short average duration of life;

for example, there can be no difference in the healthiness of two localities in one of which the death-rate is twice or even thrice as high as that of the other, provided other conditions are such that in both cases the inhabitants attain the same mean age; or conversely, supposing different populations to enjoy the same mean duration of life, any differences which may be presented by their respective death-rates are due to other circumstances than differences of health. From the death-rate alone, however, it is impossible to arrive at any definite conclusion in regard to the average duration of life. But the birth-rate and the death-rate together supply us with the necessary data. The primary object, therefore, which I set before myself was the determination of the mean duration of life under various conditions of the birth-rate and death-rate.

For the purposes of my calculations I have assumed the simplest possible condition of things; namely, 1st, that in each case the death-rate and birth-rate continue uniform from year to year; 2nd, that no immigration or emigration takes place, and that, consequently, any annual differences there may be in the number of the population are due simply to the excess of births over deaths or conversely; and 3rd, that every individual born into the population attains the mean age, or, in other words, that all the inhabitants die at the same age. Under these conditions there are three different cases for investigation; namely, 1st, that in which the births exceed the deaths and the population consequently increases; 2nd, that in which the births and deaths are equal, and the population consequently undergoes no change of number; and 3rd, that in which the deaths exceed the births, and the population therefore progressively diminishes.

In the first case (that in which the births exceed the deaths), the population, the births and the deaths, all increase from year to year in the same ratio by geometrical progression. And if the number of the population from which we start be represented as a unit, and the letters  $b$  and  $d$  represent respectively the birth-rate and death-rate of this unit, and  $r$  its increase for one year, or what is the same thing, the difference between  $b$  and  $d$ , the following three series will represent the annual growth of the population, of the births and of the deaths respectively:

	Population.		Births.		Deaths.
1st year	...	1	...	$b$	...
2nd year	...	$(1+r)$	...	$b(1+r)$	...
3rd year	...	$(1+r)^2$	...	$b(1+r)^2$	...
4th year	...	$(1+r)^3$	...	$b(1+r)^3$	...
5th year	...	$(1+r)^4$	...	$b(1+r)^4$	...
*		*		*	*
$n$ th year	...	$(1+r)^{n-1}$	...	$b(1+r)^{n-1}$	...

Now, according to the hypothesis with which we started, all the persons born in any one year, attaining as they do exactly the same age, will die together in the course of some future year. Thus, the births represented by the letter  $b$ , occurring in the first year of the series, will die in a lump, so to speak, at the end of some definite period, and those of the next year represented by  $b(1+r)$  will die similarly in the course of the year next following, and so on continuously; and it is obvious, therefore, that if we can ascertain the number of terms of the series which intervene between these respective epochs of birth and death, we shall thereby ascertain the mean duration of life. Now, since the index of  $(1+r)$  is 1 at the expiration of one year from the time at which the births,  $b$ , occurred, 2 at the expiration of two years, 3 at the expiration of three years, and  $n$  at the expiration of  $n$  years, it follows that the index of that term of the third of the above series which equals  $b$  represents the mean duration of life in years.

Let  $x$  be that unknown index the value of which it is required to ascertain. Then it is obvious that,

$$1 : (1+r)^x :: d :: d(1+r)^x$$

but by the hypothesis

$$d(1+r)^x = b;$$

so that the proportion becomes

$$1 : (1+r)^x :: d : b;$$

and, consequently,

$$(1+r)^x = \frac{b}{d}, \text{ or } x = \frac{\log \frac{b}{d}}{\log (1+r)}.$$

a formula from which the value of  $x$  can be readily deduced.

For the second case (that in which the birth-rates and death-rates are exactly equal) the above formula is useless;

for here  $b$  and  $d$  being equal to one another,  $\frac{b}{d} = 1$ ,  $r = 0$ ,

and the equation consequently becomes  $x = \frac{\log. 1}{\log. 1} = \frac{0}{0}$ , the value of which is indeterminate. The mean duration of life, however, is readily ascertainable by another process. In this case the whole number of persons supposed to be living at any one time is made up of a series of successive annual increments, which are due to the successive annual births and are equal to one another; and it is obvious that the term of the last added of these increments to die will arrive only after all the other increments have been successively removed. And hence the mean duration of life will be the quotient obtained by dividing the population by the annual increment of births.

In the third case (that, namely, in which the annual deaths exceed the annual births, and the population has a consequent tendency to die out) the formula which was made use of for the first case is again applicable; only since  $b$  is now less than  $d$ ,  $b-d$  or  $r$  becomes a minus quantity, and the ratio less than unity, so that the series (whether of population, births or deaths) which is a diminishing one is infinite, and the population, though constantly decreasing, never comes to an end.

In Table I, I have arranged the calculations which I have made (in accordance with the principles above enunciated) in regard to the combined relation of the death-rate and birth-rate to the duration of life. I have in eight successive vertical columns, in which it is assumed that the mortality is .5, 1, 1.5, 2, 2.5, 3, 3.5 and 4 respectively per cent., arranged the mean durations of life which result from a combination of these several mortuary rates and of birth-rates varying from a little over 100 per cent. to zero. The series of birth-rates which corresponds to each death-rate is placed in a vertical column to the left-hand side of the death-rate column; and all the birth-rates are so arranged that the differences between the birth-rates and death-rates on the same horizontal line of the general table are in all cases identical. These differences, which represent the annual differences in the population per cent. and range from 100 to zero (at which

latter point the births and deaths balance each other), and from zero to —3·95, occupy a special column on the extreme left of the table.

The first series of results (and these I shall quote at length) are those which are deduced from a death-rate of .5 per cent. With a birth-rate of 100·5 per cent. and an annual increase, therefore, of 100 per cent., the mean duration of life is 7·6 years; with a birth-rate of 10·5 and an increase-rate of 10 the mean duration of life is 31·9; with a birth-rate of 8·5 and an increase-rate of 8 the mean duration of life is 36·81; with a birth-rate of 5·5 and an increase-rate of 5 the mean duration of life is 49·15; with a birth-rate of 5 and an increase-rate of 4·5 the mean duration of life is 52·31; with a birth-rate of 4·5 and an increase-rate of 4 the mean duration of life is 56·02; with a birth-rate of 4 and an increase-rate of 3·5 the mean duration of life is 60·4; with a birth-rate of 3·5 and an increase-rate of 3 the mean duration of life is 65·8; with a birth-rate of 3 and an increase-rate of 2·5 the mean duration of life is 72·56; with a birth-rate of 2·5 and an increase-rate of 2 the mean duration of life is 81·27; with a birth-rate of 2 and an increase-rate of 1·5 the mean duration of life is 93·1; with a birth-rate of 1·5 and an increase-rate of 1 the mean duration of life is 110·4; with a birth-rate of 1 and an increase-rate of .5 the mean duration of life is 188·98; and with a birth-rate of .5 and a consequent stationary condition of the population the mean duration of life is 200. Lastly, with a birth-rate of .05 per cent., or of only 5 in 10,000, and a consequent annual diminution of the population by .45 per cent., or by 45 in 10,000, the mean duration of life is 510·54 years.

Now, it will be readily perceived from the above figures that under the same death-rate, namely, .5 per cent., the average duration of life progressively diminishes as the birth-rate increases, and conversely progressively increases as the birth-rate diminishes. And it is obvious as well from an inspection of the table as from a consideration of the principles on which it has been constructed, that if the annual increase of the population be conceived to increase to infinity, the mean duration of life would sink to zero; and that, on the

other hand, if the birth-rate sink to nil, the mean duration of life becomes infinite.

The second series of results are those which are obtained with a death-rate of 1 per cent. In this case the mean duration of life varies from  $6\frac{1}{3}$  years with a birth-rate of 101 and an annual increase of 100, to 100 years when the birth-rate is 1 per cent. and the population consequently neither increases nor diminishes; but when the birth-rate sinks to .5 per cent., and the population decreases at the same rate, the duration of life becomes 138.28 years; and when the birth-rate sinks to .005 per cent., and the population decreases by .95, the duration of life becomes 313.84.

In the third series of results, which are derived under a death-rate of 1.5 per cent., the duration of life appears to be 6.08 years when the birth-rate is 101.5 and the increase-rate 100, and  $66\frac{2}{3}$  years when the death-rate and birth-rate are equal and the population is stationary. With a birth-rate of .5 per cent. and an annual diminution of 1 per cent., the duration of life mounts to 109.31, and with a birth-rate of .05 per cent. and an annual diminution of 1.45 per cent. the duration of life mounts to 232.86.

The fourth series of results are connected with a death-rate of 2 per cent. Here the mean duration of life ranges from 5.67 years with a birth-rate of 102 and an increase-rate of 100, to 50 years with a birth-rate of 2 and a stationary population. With a birth-rate of .5 per cent. and an annual diminution of 1.5 the mean duration of life becomes 91.72; with a birth-rate of .05 and a diminution of 1.95, 187.32 years.

In the fifth series the death-rate is 2.5 per cent., and the mean duration of life rises from 5.85 years when the birth-rate is 102.5 and the increase-rate 100, to 40 when the birth-rate and death-rate are equal. With a birth-rate of .5 per cent. and an annual diminution of the population by 2 per cent. the duration of life becomes 79 $\frac{1}{3}$ ; and with a birth-rate of .05 and an annual diminution of 2.45, 157.71.

In the sixth series, with a death-rate of 3 per cent., the mean duration of life is 5.10 years with a birth-rate of 103 and an increase-rate of 100, and 38 $\frac{1}{3}$  when the birth-rate and death-rate are equal. When the birth-rate is .5 per cent., and the annual decrease of the population is 2.5 per cent., the

duration of life is 70.77 years; and when the birth-rate is .05 and the decrease is 2.95 the duration of life is 136.73.

The seventh series is dependent on a death-rate of 3.5 per cent. In this case the mean length of life ranges from 4.89 years when the birth-rate is 103.5 per cent. and the increase-rate 100, to 28.57 years when the birth-rate and death-rate are equal to one another. The duration of life increases to 63.89 years when the birth-rate is only .5 per cent. and the population diminishes by 8 per cent. per annum, and to 121.01 when the birth-rate becomes .05 and the annual diminution per cent. 3.45.

In the last series it is assumed that the death-rate is 4 per cent. Here with a birth-rate of 104, and an increase-rate of 100, the mean duration of life is 4.70 years; and with a birth-rate of .4 per cent. and a stationary population 25 years. With a birth-rate of .5 per cent. and an annual loss of 3.5 per cent. of the population the duration of life mounts up to 58.37 years; while with a birth-rate of .05 and an annual loss of 3.95 it rises to 108.73.

It may be added that in all cases, as well as in the first, the increase of the birth-rate to infinity reduces the mean duration of life to zero, and the reduction of the birth-rate to zero raises the mean duration of life to infinity.

The table shows further the degree in which, under the same rates of increase, the mean duration of life varies as the death-rate varies. Thus, with an increase of 100 per cent. the mean duration of life, which is 7.6 when the death-rate is .5 per cent., becomes successively 6.66, 6.08, 5.67, 5.35, 5.10, 4.89, and 4.7, as the death-rate increases by increments of .5 up to 4 per cent.; and with a stationary population, under similar conditions of the death-rate, the duration of life is 200, 100, 66 $\frac{2}{3}$ , 50, 40, 38 $\frac{1}{3}$ , 28.57, and 25.

The table shows also the degree in which, under the same birth-rates, the mean duration of life becomes shorter as the death-rate increases. Thus, assuming the birth-rate to be 4 per cent., the average duration of life becomes, with a death-rate of .5 per cent., 60.4 years; with a death-rate of 1 per cent. 46.9; with a death-rate of 1.5, 39.74; with a death-rate of 2, 35; with a death-rate of 2.5, 31.56; with a death-rate of 3, 28.91; with a death-rate of 3.5, 26.77; and with a death-rate

of 4, 25 ; and assuming a birth-rate of .05 per cent., or of 5 in 10,000, the mean length of life becomes for the same death-rates, taken in the same order, 510.54 years, 313.84, 232.86, 187.32, 157.71, 136.73, 121.01, and 108.73.

But the tables further demonstrate clearly, and this is the point of chief importance, that the health of a population as measured by the duration of life may under certain conditions of the birth-rate be as good when the mortality is high as when the mortality is low. Thus, the mean duration of life is the same, namely 81 years, with a death-rate of .5 per cent. and a birth-rate of 2.5 per cent., as it is with a death-rate of 1 per cent. and a birth-rate of 1.5 per cent., or with a death-rate of 1.5 per cent. and a birth-rate of 1 per cent. ; it is the same, namely 37 years, with a death-rate of 1.5 per cent. and a birth-rate of 4.5 per cent., as it is with a death-rate of 2 per cent. and a birth-rate of 3.5 per cent., or a death-rate of 3.5 per cent. and a birth-rate of 2 per cent. ; it is the same, namely 31 years, with a death-rate of 1.5 and a birth-rate of 6, as it is with a death-rate of 2 and a birth-rate of 5, or with a death-rate of 3 and a birth-rate of 3.5, or with a death-rate of 3.5 and a birth-rate of 3, or with a death-rate of 4 and a birth-rate of 2.5 ; it is the same, namely 58 years, with a death-rate of .5 and a birth-rate of between 4 and 4.5 per cent., as it is with a death-rate of 4 and a birth-rate of .5. Other examples might be adduced from the table, but it is unnecessary, inasmuch as a little consideration will render it apparent that by interpolating other birth-rates and mean durations of life between those given in the table we may readily find exactly corresponding terms under each death-rate column.

It may be objected to my table that it comprises amongst conditions which are quite possible other conditions which are impossible and absurd : that, for example, a birth-rate of 100 per cent. could only be maintained in a population of which every female bore annual twins from the time of her birth to that of her death, while even a birth-rate of 10 per cent. is scarcely compatible with any possible conditions of a population ; and that, on the other hand, not only is it absolutely certain that the age of 200 will never be reached by human beings, but that the attainment of a mean duration of life of even 70 years is entirely beyond the anticipations of

sober-minded sanitarians. The answer to these objections is, of course, that the table has been constructed primarily with the object of showing clearly the mutual relations of the birth- and death-rates, and that these are most strikingly exhibited by adducing extreme cases. Yet what holds good of extreme cases holds good in principle of all other cases; and to render the table practically useful it is only necessary to disregard the former, and to limit our attention to the latter.

The highest birth-rate which I see recorded in the Registrar General's annual summary, for 1875, is that of Gateshead, which was 4.76 per cent. The lowest is 2.42 and occurred at Cheltenham. Now there is no physical impossibility in the reduction of the birth-rate considerably below that recorded of Cheltenham; it is conceivable, indeed, that under special conditions it might fall to zero. But, on the other hand, though the birth-rate might easily rise above that of Gateshead, the number of children capable of being born *per annum* must be limited by the reproductive capacity of those members of the female population who are of child-bearing age. This age extends from puberty to 45, or in a well-ordered population may be taken to extend practically from 20 to 45. With the object of arriving at some kind of notion of the possible reproductive capacities of populations existing under the several conditions which I have assumed for the purposes of Table I, I have calculated (see Table II) the percentage number of persons between 20 and 45 years of age living under each several combination of the birth- and death-rates. Now, if males and females existed in equal numbers, and every woman gave birth to a child once in two years, the fourth part of each number arrived at should give the highest possible number of births per cent. *per annum* attainable in the case to which it relates. Hence it will be readily seen that a birth-rate of 6 per cent. might be maintained in such a population with a death-rate of either .5 or 1 per cent.; a birth-rate of 5.5 per cent. in a population dying at the rate of 1.5 or 2 per cent.; a birth-rate of 5 per cent. in a population dying at the rate of 2.5 per cent.; a birth-rate of 4.5 per cent. in a population dying at the rate of 3 per cent.; and a birth-rate of 4 per cent. in populations dying at the rate of 3.5 and 4 per cent.

It is obvious, of course, that these calculations do not represent anything that would actually obtain under the continuance of the combined birth- and death-rates to which they severally relate ; for we know that females and males do not exist in equal proportion ; that not all women marry ; and that of those who marry some marry early, some late, some have no children, some but few, others large families ; and we know further that the mean age the attainment of which was an essential element in my calculations does not represent the actual age which all persons reach, but that in contributing to maintain it many persons live beyond it, many fall short of it. While, however, the former considerations would tend to reduce the possible increase of populations by births below my estimates, the last demonstrates that even in those cases in which the mean duration of life is under 20, and in which, therefore, on my original assumption there is little or no reproductive material, there would be actually living a considerable number of women above the age of 20, and that generally in the series of cases which I have just quoted there would certainly be a considerably larger percentage of adult population than my figures show. At any rate, the practical conditions which tend to reduce the possible birth-rates under the assumed relations of the birth- and death-rates, and those which tend to increase them, balance one another to some extent and thus tend to confirm the possibility of the conclusions to which my calculations lead.

I have drawn in each column of Table I a horizontal line immediately above the highest birth-rate which, on the grounds above given, seems to be reasonably attainable in connection with the death-rate belonging to that column.

The mean duration of life in England has been calculated to be nearly 41 years. It is impossible to predict to what extent sanitary science will ultimately succeed in raising it. It is certain, however, that its rise must depend, less on increasing the duration of life beyond what is commonly regarded as the normal span, namely 70 or 80 years, than on the prevention of deaths in the earlier periods of life, and especially in early infancy. If we assume that, in consequence of the prevention of premature mortality, the mean duration of life is raised to 70 years, we shall find that in the first column of Table I

the conditions become impossible when the birth-rate falls below 3.5 per cent.; in the second column, when the birth-rate falls below 2 per cent.; in the third column, when the birth-rate falls below 1.5 per cent.; in the fourth, fifth, and sixth columns, when the birth-rate falls below 1 per cent.; and in the seventh and eighth columns, when the birth-rate falls below .5 per cent. Beneath these several birth-rates and their associated estimates of the duration of life I have again drawn horizontal lines; so that the conditions which may be regarded as possible conditions are in each column included between the upper and lower horizontal lines. It may be added that the apparent irregularities in the table presented by the upper and lower limits of the assumed possible conditions are due simply to the small number of birth-rates which have been employed; and that it would be easy, if it were worth while, to ascertain for each column, on the one hand, the exact birth-rate which would allow of a mean duration of life of 70 years, and, on the other hand, the exact birth-rate which in accordance with my assumptions is compatible with the lowest possible mean duration of life. It may also be added (what is, however, sufficiently obvious) that a population in which there are no births must gradually die out, and that it is impossible in such a case that a uniform annual death-rate can be maintained. Indeed, however healthy and long-lived such a population might be, and however low its initial death-rate, the death-rate must of necessity increase year by year until it becomes with the extinction of the population cent. per cent.

In calculating the mean durations of life given in Table I, I assumed (as will be recollected) that every person born into a population attains the same age, and further that the children born in a group (so to speak) in one year, die in a group in the course of some subsequent year. But the truth of the table is not dependent on these assumptions. The results as regards the duration of life in relation to uniformly maintained birth-rates and death-rates are in fact identical, whether the same length of life be attained by every one of the inhabitants, or whether as occurs in nature the mean duration be the resultant of lives varying widely among themselves in length. And again, in order to determine the mean duration of life, it is not really necessary to assume that persons are born in

groups and die in groups. For if we take as the basis of our calculation just so much of a population as yields one birth and take  $d$  to be the mortality due to the same fragment of the population, the duration of life will be determined by that index of  $(1+r)$  which makes  $d (1+r)^x = 1$ ; and the equation becomes  $x = \frac{\log \frac{1}{d}}{\log (1+r)}$ .

For the sake of illustrating the use of the foregoing calculations, and at the same time the errors to which a blind reliance upon them might lead, I will adduce and discuss some of the statistics relating to the population of Camberwell, the parish with which I am more particularly connected.

In Table III, I have tabulated for the whole parish and for its four sub-districts (namely Dulwich, Camberwell, Peckham, and St. George's) the populations as determined by the censuses of 1861 and 1871, together with the total number of births, the total increase from immigration, the total addition to the population from these combined sources, the actual increase of the population, and the total number of deaths, occurring in the ten years included between these epochs.

In Table IV, I have given, for the same parish and sub-districts, not the actual number of births, immigrants, and deaths which accrued during the first year of the above decenniad (for the second of these items could not be ascertained), but the number of births, immigrants, and deaths, which would have accrued in that year if their respective totals for the ten years had been the result of an annual increase in geometrical ratio. I have incorporated also in the table the total additions due to births and immigration, and the estimated increase of the several populations, all for the same year.

In Table V, I have calculated, from the above data, the percentage birth-rates, immigration-rates, combined birth- and immigration-rates, death-rates and increase-rates which may be assumed to have prevailed in the parish and in its subdivisions during each of the ten years included in the period under consideration.

It will be seen from this that the birth-rate per cent. was about 4 in St. George's, about 3.6 in Peckham and in the

parish as a whole, 3.2 in the Camberwell sub-district, and only 2 in Dulwich; and that the death-rate per cent. was in St. George's 2.4, in Peckham 2.3, in the whole parish 2.2, in Camberwell 1.9, and in Dulwich 1.3.

According to these data the mean duration of life should be (see Table VI), for Dulwich 60.07 years, for Camberwell 40.03, for the whole parish 35.6, for Peckham 34.36, and for St. George's 32.33. And (it need scarcely be added) these figures are in some sort of relation, not only with the respective death-rates of these several localities, but with what we know on other grounds of the conditions of life and health in them. It may be pointed out that if (other things being equal) the birth-rate in Dulwich had been as high as that of St. George's, the estimated mean duration of life would have been 42.35 instead of 60.07; and that if the birth-rate of St. George's had been as low as that of Dulwich, the estimated duration of life would have been 44.43 instead of 32.33.

It will be observed that in these last calculations I have made no distinct reference to the immigration which has been taking place in varying proportions into the different parts of the parish. I have assumed, in fact, as is I believe generally assumed, that the added population resembles the original population in all its vital attributes; that the death-rate and birth-rate, and consequent rate of increase, are the same in both cases. There is, however, another mode of regarding the immigrants: it is to look upon them as births into the parish, and to estimate their duration of life, not from the time of their actual birth, but from the moment at which they became parishioners. On the original assumption, the mean duration of life of immigrants would of course be exactly equal to that of the natives; but on the last the estimated mean duration of life of the immigrants would be simply the mean duration of their residence in the parish.

A calculation made on the assumption that the increase of population of the parish and of its parts, actually due to births and immigration, is due to births alone, reduces the estimated mean duration of life in the whole parish from 35.6 to 25.47, in Dulwich from 60.07 to 24.79, in Camberwell from 40.03 to 29.17, in Peckham from 34.36 to 25.34, and in

St. George's from 32.83 to 28.0;<sup>1</sup> and since these reduced estimates are necessarily, each, a mean between proportional numbers of persons actually added by birth to the population and spending the whole of their life in the parish, and of immigrants spending only the latter portion of their life in the parish, we arrive by an easy calculation at the result, that for the whole parish the immigrants became resident at 21.92 years of age and lived 13.68 years subsequently, that for Dulwich they became resident at 44.99 years of age and survived 15.08 years, that for Camberwell they became resident at 29.5 years of age and survived 10.53 years, that for Peckham they became resident at 20.8 years of age and survived 13.56 years, and that for St. George's they became resident at 22.46 years of age and survived 9.87 years.

It will probably be remarked that the above results by no means represent the actual condition of things prevailing in Camberwell, that (to take an extreme example) the mean duration of life in Dulwich is unquestionably less than 60 years, and that the average age of immigrants into that district is certainly not 45 years. All this I fully admit, but I maintain that the results would have been much more inaccurate and untrustworthy if the births had been disregarded as an element in the calculation. The fact, of course, is, that in dealing with vital statistics many matters must be taken into consideration besides the births and deaths and mere number of the population, and many of these are obscure and incapable of numerical expression. I will conclude my paper by considering a few of these.

1st. I have assumed throughout my calculations that the growth of the population has been in geometrical progression. So far as its growth by means of births is concerned the assumption is no doubt fairly accurate, but it is certainly very far from accurate as regards that portion of its growth which is due to immigration. The circumstances which determine migration are various and variable in a high degree; and

<sup>1</sup> Since writing this paragraph I have satisfied myself that the latter part of it involves a fallacy. It would take too long to discuss it here; but it is connected with the fact that the estimation of the mean duration of life, where we have to deal with both immigrants and births, is a much more complicated matter than I at first supposed. I have allowed it to stand, however, as an example of the errors adverted to on page 255.

though they may combine to produce some orderly result in the case of large populations like that of the whole metropolis, it is certainly not so with regard to small populations like those of parishes and districts. It is much more probable that in the case of Dulwich the immigration-increase has been by arithmetical than by geometrical progression; and it is obvious that, if this be so, my results in reference to the vital statistics of immigrants are not trustworthy.

2nd. It has been taken for granted that the added populations resemble the original populations in all their conditions, especially in their birth-rate, death-rate, and average age. But this is an assumption which, though hard to dispute, is probably erroneous. And if, for example, as might well be the case, either the birth-rate or the death-rate, or both, were a little higher among the natives of Dulwich than among the immigrants, the calculated mean duration of life of the natives would fall to a more reasonable figure, and as a consequence the estimated age of immigrants would also fall more or less considerably below my present impossible estimate. The same remarks apply equally to the other districts.

3rd. In speaking of a population and of immigrants, I have assumed that all who come into a locality remain in it and die in it. It is a fact, however, of the utmost importance, that emigration as well as immigration is constantly going on, and that consequently our statistical units of population do not represent actual individuals, but consist in a greater or less degree of blended fragments, so to speak, of several lives. Thus, to take a common and simple example from many of a somewhat similar kind. A resident family comprises the father, the mother, several children, and three or four servants. Now, the servants leave from time to time and are replaced by others, each statistical life being continued, so to speak, from the servant that leaves to her successor, and so on continuously. The children also migrate after awhile, some probably leaving the district. It is obvious that here again each one of these (so far as the district is concerned) uncompleted lives become statistically incorporated with some immigrant life.

Now it is clear that such statistically continuous and almost immortal lives must by their presence reduce the death-rate

and increase the mean duration of life ; and that if they be present in large proportion their influence in this respect must be very great. It is certain that the low death-rate and high mean length of life in Dulwich are largely attributable to this cause. It need scarcely be added, on the other hand, that if the lives which replace others or are added to the population are inferior in health, as occurs when a hospital or infirmary attracts patients, or when those who have left come home to die, the death-rate of the population becomes increased and the average duration of life diminished.

In conclusion, and as bearing upon the questions which have just been discussed, I venture to quote some remarks which I addressed to the vestry of Camberwell in my report for the year 1863. They are subject, of course, to correction in reference to the influence of the birth-rate on the significance of the death-rate, which at that time I overlooked.

“ But supposing a death-rate to have been ascertained with perfect accuracy ; what does it teach ? what do we learn from it ? The prime fact, of course, is that out of so many persons living, so many persons have died in a given time. Experience has shown that in ordinary years death-rates vary within comparatively narrow limits ; that in the presence of severe outbreaks of epidemic diseases death-rates become augmented, and often very seriously augmented ; that in certain localities (such as large towns) which are generally assumed to be of inferior healthiness the death-rates are mostly higher than they are in certain other localities (such as country districts) which enjoy the reputation of comparative salubrity. And on these and other grounds it has been assumed that the death-rate is a measure of the health of a population, and that the fluctuations of the death-rate imply corresponding fluctuations of health. To a certain extent this is true ; but it is true only of the margin which remains after *necessary* deaths (if I may use the expression) have been eliminated (and these constitute the great bulk of a mortality) ; and after the effects of all influences tending to disturb the due proportion between the young, the middle-aged, and the old, the healthy, the sick, and the dying have been investigated and allowed for. On the effects of those deaths which I have termed necessary upon death-rates I need not now enlarge.”

"On the effects of the second class of causes, however, I have something to say, especially as I believe that they have a very important influence, and that this influence has been very generally overlooked. The influence of hospitals, of asylums, and of workhouses and of any institution receiving the aged and sick is well illustrated in the present report, by the excessive mortality which the presence of the workhouse and of two lunatic asylums gives to the subdistrict of Camberwell. But it may happen in another way that, by a kind of process of natural selection, the young and able-bodied tend to accumulate in certain localities, while the infants and the aged, and it may be the sickly, remain in undue proportion in other localities. To illustrate this: the death-rate of Dulwich has always been remarkably low; the death-rate of Peckham, without being *really* remarkably high, has always been *by contrast* remarkably high, and it has been assumed that therefore Dulwich is far more healthy than Peckham. I believe Dulwich to be very healthy; I know that Peckham, in its low-lying parts, has many elements of unhealthiness; but I do not for one moment believe that the difference of their death-rates indicates a corresponding difference in their healthiness, still less, of course, that the excess of its death-rate is a measure of the degree in which diseases, preventable by the action of the vestry, prevail in Peckham. I have said on former occasions, and I repeat it now, that there is in many parts of Peckham a large poor population; that amongst such a population children get neglected and die of illnesses, from which under other conditions they would doubtless have recovered; that from their habits of life contagious diseases spread among them with far greater virulence than they do among the so-called better classes, and that illnesses are manufactured amongst them which those who live in different circumstances rarely see. I have said formerly, and I say again, that (although important improvements have been made) there are many localities in Peckham which, in great measure from want of sewerage, are still in a condition to make them (in the presence of epidemics of cholera or of fever) hotbeds of these diseases. Fortunately we have escaped such disasters. Doubtless some of these conditions have influenced injuriously the death-rate of Peckham. But Dul-

wich comprises mainly the houses of the rich, Peckham mainly the houses of the poor. A typical Dulwich household consists of the father, mother and children, and of a considerable number of other persons—domestic servants—in the prime of life, and for the most part in the prime of health, draughted, so to speak, from the poorer households and therefore from the poorer localities. Now, the mortality in such a household is as a rule the mortality of the family, and not the mortality of the servants of the family. If the latter become ill and die, their deaths rarely if ever occur in the houses to which they are attached; their own homes or the hospital become their final refuge. So that in such a household there is, in varying but often large proportion, a population which can add nothing to the deaths of the household, and the presence of which, in a statistical sense, must necessarily therefore produce a fallacious appearance of healthiness—a condition the exact converse of that which obtains at a workhouse or a hospital. Obviously Dulwich, which comprises such households in large proportion, owes no inconsiderable proportion of its apparent healthiness to this cause. Peckham, on the other hand, comprises in large measure the houses of the poor. Their inmates consist of the parents and a family of children, but of no domestic servants. In fact, the elder and adult children are just the material of which domestic servants are made, and have probably quitted home for service elsewhere. So that in such a household not only are the numbers not swelled by the influx of a (so to speak) non-dying population, but on the contrary are diminished by the efflux of a certain number of those who, in a statistical sense, are the very vitality of the family. There remains, therefore, a population somewhat below the normal average in health and strength, which of itself would naturally yield a comparatively high death-rate, and in which the death-rate is liable to be still further increased, from the fact that many of those who have left the parent roof return thither to die when fatal illness overtakes them. I am not, of course, asserting that all the households of Peckham are of one class, or that all the households of Dulwich are of another class; I am not asserting that all the elder children of the poor go out to service, or that of those that die all die at their own homes and none in

the house of their master. But I assert that the one class of household prevails in Peckham, the other class prevails in Dulwich; that the differences in regard to them which I have pointed out are real differences, which must necessarily affect the respective death-rates of Peckham and of Dulwich; and that differences of death-rate due to these conditions are totally unconnected with local salubrity or insalubrity."

"The fact is that the sanitary condition of a locality is much more truly measured by the comparative prevalence in it of certain forms of disease than by mere variations of death-rate; although no doubt a large, and especially an increasing, death-rate is a matter to arrest attention and to demand inquiry. Thus if, in a certain place, ague prevails, we know it to be unhealthy from the presence of marsh miasm; if cholera or typhoid fever prevails in a locality, we have good reason to believe that in that locality the atmosphere or the water is unduly impregnated with the poison of cesspools or of sewers; if typhus fever breaks out, there is ground to suspect that the population among whom it appears are overcrowded, their houses filthy and insufficiently ventilated. But it does not necessarily follow that the presence or the spread of such diseases proves local insalubrity; many of them are infectious, and if introduced into a crowded neighbourhood will spread in it however otherwise healthy that neighbourhood may be. The prevalence of any of them, however, in a markedly aggravated form, or for an unusually long period, renders the presence of local causes of insalubrity exceedingly probable."

The subject of the effects of migration in disturbing local rates of mortality has since been very laboriously and ably investigated by Mr. Welton, whose results agreeing with but extending my own, as given in the report just quoted, were announced by him in a paper read before the Institute of Actuaries December 19th, 1870, and published in the 16th volume of their Journal. Let me commend that valuable paper to the notice of all who are interested in the subject.

## Birth-rate and Death-rate.

Annual variation of rent per cent.	Mean duration of life.	Death-rate 8 per cent.		Death-rate 8·5 per cent.		Death-rate 9 per cent.	
		Birth-rate per cent.	Mean duration of life.	Birth-rate per cent.	Mean duration of life.	Birth-rate per cent.	Mean duration of life.
Increasing 100	5	103	5·10	103·5	4·89	104	4·70
10	8	13	15·38	13·5	14·16	14	13·14
8	5	11	16·88	11·5	15·46	12	14·28
5	2	8	20·10	8·5	18·19	9	16·62
4·5	9	7·5	20·82	8	18·78	8·5	17·12
4	6	7	21·60	7·5	19·43	8	17·67
3·5	5	6·5	22·47	7	20·15	7·5	18·27
3	7	6	23·45	6·5	20·94	7	18·93
2·5	7	5·5	24·54	6	21·83	6·5	19·66
2	8	5	25·80	5·5	22·82	6	20·47
1·5	6	4·5	27·23	5	23·96	5·5	21·39
1	4	28·91		4·5	25·26	5	22·43
0·5	2	8·5	30·91	4	26·77	4·5	23·61
Stationary 0		8	33·33	8·5	28·57	4	25
Diminishing	0·45						
	0·5	—	—	—	—	—	—
—	0·95	2	2·5	86·87	8	30·75	8·5
—	1	—	—	—	—	—	—
—	1·45	3	2	40·34	2·5	33·48	3
—	1·5	—	—	—	—	—	—
—	1·95	3	1·5	45·86	2	37·03	2·5
—	2	—	—	—	—	—	—
—	2·45	6	1	54·98	1·5	41·94	2
—	2·5	1	—	—	—	—	—
—	2·95	—	0·5	70·77	1	49·47	1·5
—	3	—	0·05	136·78	—	—	—
—	3·45	0	∞	0·5	63·89	1	45·52
—	3·5	—	—	0·05	121·01	—	—
—	3·95	—	—	0	∞	0·5	58·37
—	4	—	—	—	—	0·05	108·78
—	—	—	—	—	—	0	∞



TABLE II.—Showing Number of Persons living per cent. between 20 and 45, under different conditions of Birth- and Death-rates.

Annual rate of population per cent.	Birth-rate per cent.	Death-rate, 1 per cent.	Death-rate, 1.5 per cent.	Death-rate, 2 per cent.	Death-rate, 2.5 per cent.	Death-rate, 3 per cent.	Death-rate, 4 per cent.	
							Persons living between 20 and 45, per cent.	
							Persons living between 30 and 45, per cent.	Birth-rate, per cent.
100	100.5	—	101	—	101.5	—	102	102.5
10	10.5	10.54	11	6.35	11.5	2.09	12	12.5
5	5.5	29.22	6	26.22	6.5	18.93	7	12.74
4	4.5	32.08	5	32.04	5.5	25.28	6	18.48
3.5	4	38.08	4.5	36.61	5	28.72	5.5	21.84
3	3.5	38.73	4	38.57	4.5	33.03	5	25.59
2.5	3	33.73	3.5	33.35	4	37.69	4.5	29.92
2	2.5	32.85	3	39.35	3.5	49.75	4	34.58
1.5	2	30.75	2.5	38.45	3	46.15	3.5	39.11
1	1.5	27.07	2	36.72	2.5	45.12	3	45.86
0.5	1	31.22	1.5	31.84	2	42.42	2.5	52.56
0	0.5	12.5	1	25.0	1.5	86.5	2	50.0

The formulae used in calculating the above table are as follows:

$$s = \text{proportional number of persons living from 20 years of age upwards.}$$

$$s_1 = \text{proportional number of persons living between 20 and 45 years of age.}$$

$$d = \text{death-rate of one unit per annum.}$$

$$r = \text{annual increase of one unit.}$$

$$n_1 = \text{duration of life subsequent to 20 years of age.}$$

The result has to be multiplied by 100.

1. For those cases in which the mean duration of life lies between 20 and 45.  
 2. For those in which the mean duration of life is over 45.

$$s = \frac{d}{r} \left\{ \frac{(1+r)^{n_1} - 1}{(1+r)^{n_1} - 1} \right\}$$

$$s_1 = \frac{d}{r} \left\{ \frac{(1+r)^{n_1} - 1}{(1+r)^{n_1} - 1} \right\}$$

1 This table was made before Table I was completed; and it was not thought worth while to extend it in conformity with that table.

TABLE III.—*Statistical facts relating to Population of Camberwell for the ten years 1861-71.*

	Whole Parish.	Dulwich.	Camberwell	Peckham.	St. George's.
Population in 1861 . . .	71,488	1723	21,297	28,135	20,333
Population in 1871 . . .	111,302	4041	31,251	42,159	33,851
Births, 1861-71 . . .	32,043	576	8,360	12,531	10,576
Immigrants, 1861-71 . . .	27,588	2094	6,578	9,597	9,269
Total additions 1861-71 . . .	59,581	2670	14,988	22,128	19,845
Deaths, 1861-71 . . .	19,767	352	4,984	8,104	6,827
Increase, 1861-71 . . .	39,814	2318	9,954	14,024	13,518

TABLE IV.—*Estimated<sup>1</sup> Births, Immigrants, Deaths, &c., for the year 1861-62.*

	Whole Parish.	Dulwich.	Camberwell.	Peckham.	St. George's.
Births . . .	2604·405	38·09189	699·2582	1087·5770	831·8756
Immigrants . . .	2238·245	138·4799	550·2058	794·6395	729·0712
Total additions . . .	4842·650	176·5718	1249·464	1832·217	1560·947
Deaths . . .	1606·631	23·27888	416·8783	671·0181	497·6624
Increase . . .	3236·019	153·29342	832·5857	1161·1989	1063·2846

<sup>1</sup> Formula employed in these calculations:—

$s$  = sum of series (total births, deaths, or the like for 10 years).

$$a = \frac{sr}{(1+r)^{10} - 1}$$

$a$  = first term.

$r$  = increase of 1 per annum.

TABLE V.—*Birth-, Immigrant-, Death-, and other rates per cent. per annum.*<sup>1</sup>

	Whole Parish.	Dulwich.	Camberwell.	Peckham.	St. George's.
Birth-rate . . .	3.562505	2.116619	3.220417	3.613287	3.987010
Immigrant-rate . .	3.061644	7.694793	2.538959	2.767276	3.494288
Combined-rate . .	6.624148	9.811413	5.754375	6.380566	7.481298
Death-rate . . .	2.197678	1.293484	1.919923	2.336771	2.385193
Increase-rate determined from same data as other rates. . .	4.42647	8.51792	3.88445	4.0437	5.09610
Increase-rate determined directly from censuses . .	4.52664	8.89810	3.90933	4.1272	5.22988

<sup>1</sup> Formula employed in these calculations:—

$p$  = population at end of first year.

$a$  = first term (births, deaths, or the like for 1861-62).

$r$  = increase of 1 per annum.

$$\text{rate} = \frac{100 a}{p + p r}$$

$$\frac{2}{}$$

TABLE VI.—*Mean Duration of Life under various aspects.*

	Whole Parish.	Dulwich.	Camberwell.	Peckham.	St. George's.
Mean duration of life as deduced from birth- and death-rates . . . .	35.6	60.07	40.08	34.36	32.33
Mean duration of life as deduced from combined-, immigrant-, and birth-rates, &c. .	25.47	24.79	29.17	25.84	23.0
Mean duration of life of immigrants in parish <sup>2</sup> . . . .	18.68	15.08	10.53	18.56	9.87
Mean duration of life of immigrants before coming into parish <sup>2</sup> . . . .	21.92	44.99	29.50	20.80	22.46

<sup>2</sup> See note page 259.



CONTINUATION  
OF THE  
MEDICAL HISTORY OF THE CLERGY  
MUTUAL ASSURANCE SOCIETY.

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By W. H. STONE, F.R.C.P.,  
AND  
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FELLOW OF THE INSTITUTE OF ACTUARIES.

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IN the volume for 1872 of these Reports some facts and statistics were given as to the early medical history of this institution. We have reserved a second communication until the completion of another quinquennial period, and the declaration of the ninth bonus, giving fully forty-five years of experience.

Even this instalment can only be looked upon as preliminary to a fuller investigation of the valuable materials gradually collected, on the completion of our fiftieth anniversary. The importance of collating the records of death with the number of insured members at each period was insisted on in the last paper, and requires to be even more forcibly pointed out in the present. But there are many considerations, medical as well as numerical, which can be suggested by a sum total of 1135 deaths spread over forty-five years, some of which we now propose to comment on.

It may be stated in the beginning that the bulk of the

deceased insurers, to the number of 1023, have been clergymen of the Church of England; only fifty-five having been laymen, and 57 females. There seems no obvious reason for which the former of the two accessory classes should be separated from the first. The latter, that of females, presents so many marked differences in the estimated value of life and in the special characters of the risks which they run, that it undoubtedly requires separate tabulation. The total number of both is, however, relatively small, and does not give any sufficient basis for individual deductions. It has been usual in conducting the business of an office mainly clerical to separate the three classes, and the same division is preserved in the present paper, with the understanding that only the broadest facts and generalisations will be noted in respect of the two latter.

The first table gives causes of death in classes, with their numbers and ages in decennial periods, according to the classification adopted in similar inquiries by the other Life Insurance offices.

TABLE I.

Causes of Death.	Total	Age at Death.							
		Und. 20 30	20 to 29	30 to 39	40 to 49	50 to 59	60 to 69	70 to 7	80 and above
<b>CLASS I.</b>									
Smallpox .....	2	...	...	...	1	1	...	...	...
Scarlatina .....	6	...	...	2	4	...	...	...	...
Diarrhoea .....	7	...	1	...	1	2	2	1	...
Dysentery .....	4	...	...	...	1	2	1	...	...
Cholera .....	2	...	...	1	...	...	1	...	...
Influenza .....	2	...	...	...	...	...	2	...	...
Fever—Typhus.....	10	...	1	3	1	4	1	...	...
Enteric .....	14	...	1	2	3	3	4	1	...
Remittent .....	5	...	...	2	...	3	...	...	...
Unstated.....	15	...	2	4	2	6	1	...	...
Erysipelas .....	8	...	...	1	1	4	1	1	...
Pyemia .....	6	...	...	...	1	1	4	...	...
Malarious fever.....	1	...	...	...	...	1	...	...	...
Purpura .....	1	...	...	...	1	...	...	...	...
	83	...	5	15	16	27	17	3	...

TABLE I—continued.

Causes of Death.	Total	Age at Death.							
		Und. 20	20 to 39	30 to 39	40 to 49	50 to 59	60 to 69	70 to 79	80 and above
<b>CLASS II.</b>									
Dropsey .....	4	...	...	...	2	1	...	1	...
Abcess .....	1	...	...	...	...	1	1	3	3
Gangrene .....	7	...	...	2	10	11	10	4	...
Cancer .....	37	...	...	...	...	1	2	2	...
Gout .....	5	...	...	...	...	...	2	...	...
Atrophy .....	2	...	...	...	...	...	2	...	...
Anæmia .....	2	...	...	...	...	...	2	...	...
Ulceration .....	1	...	...	...	...	1	...	1	...
Progressive muscular atrophy .....	1	...	...	...	...	...	...	1	...
	60	...	...	2	12	15	20	11	...
<b>CLASS III.</b>									
Cephalitis .....	2	...	...	...	1	...	1	...	...
Apoplexy .....	92	...	1	6	13	28	26	15	3
Paralysis .....	65	...	...	2	6	15	27	14	1
Epilepsy .....	6	...	...	2	3	1	...	...	...
Cerebral .....	64	...	...	7	17	16	14	10	...
Spinal .....	1	...	...	...	...	...	...	1	...
Locomotor ataxy .....	1	...	...	...	...	...	...	1	...
Paraplegia .....	2	...	...	...	...	1	1	...	...
Meningitis .....	3	...	...	1	2	...	...	...	...
Coma .....	1	...	...	...	1	...	...	...	...
Cerebral aneurism .....	1	...	...	...	...	1	...	...	...
Delirium .....	1	...	...	1	...	...	...	...	...
Mania .....	2	...	...	1	...	...	1	...	...
Concussion .....	2	...	...	...	1	...	2	...	...
Insanity .....	1	...	...	1	...	...	...	...	...
Effusion .....	1	...	...	...	...	1	...	...	...
	245	...	1	21	43	65	71	40	4
<b>CLASS IV.</b>									
Laryngitis .....	5	...	...	1	...	...	3	1	...
Bronchitis .....	48	...	...	1	2	12	11	16	6
Pleurisy .....	3	...	...	...	1	1	1	...	...
Pneumonia .....	38	...	2	2	10	11	8	4	1
Hydrothorax .....	3	...	...	1	1	...	1	...	...
Asthma .....	2	...	...	...	...	1	1	...	...
Consumption .....	62	...	3	18	20	17	4	...	...
Emphysema .....	2	...	...	...	...	...	...	2	...
Abcess of lungs .....	1	...	...	...	...	1	...	...	...
Congestion of lungs .....	10	...	...	...	4	3	3	...	...
Thoracic tumour .....	1	...	...	...	...	...	1	...	...
Bronchiectasis .....	1	...	...	...	...	1	...	...	...
Œdema .....	1	...	...	...	...	1	...	...	...
Hæmoptysis .....	7	...	...	2	2	3	...	...	...
Disease of chest .....	1	...	...	...	1	...	...	...	...
	185	...	5	25	41	51	33	23	7

TABLE I—*continued.*

Causes of Death.	Total.	Age at Death.								
		Und. 90	90 to 29	30 to 39	40 to 49	50 to 59	60 to 69	70 to 79	80 and above	
<b>CLASS V.</b>										
Aneurism .....	7	...	...	2	4	1	...	...	...	...
Pericarditis .....	2	...	...	...	...	1	...	1	...	...
Disease of heart .....	110	...	...	3	9	30	39	26	3	
Atheroma .....	1	...	...	...	...	...	...	1	...	...
Angina .....	16	...	...	...	...	7	7	1	1	
Paralysis of heart .....	1	...	...	...	1	...	...	...	...	...
Fatty degeneration of heart .....	9	...	...	1	...	1	5	2	...	
Endocarditis .....	2	...	...	...	...	1	...	1	...	
Phlebitis .....	1	...	...	...	...	...	...	1	...	
	149	...	...	6	14	41	51	33	4	
<b>CLASS VI.</b>										
Splenic .....	1	...	...	...	...	1	...	...	...	...
Enteric and gastric .....	8	...	1	1	1	3	...	2	...	
Peritonitis .....	7	...	...	3	1	1	1	1	1	
Ulcer of bowels .....	2	...	...	...	...	1	...	1	...	
Obstruction of bowels .....	13	...	1	1	...	3	5	3	...	
Hæmatemesis .....	7	...	...	...	...	2	3	1	1	
Disease of stomach and bowels .....	8	...	...	...	1	4	3	...	...	
Hepatitis .....	4	...	1	...	1	1	1	...	...	
Jaundice and gall-stones .....	9	...	...	...	2	2	...	5	...	
Disease of liver .....	27	...	6	4	10	6	6	1	...	
Disease of oesophagus .....	1	...	...	...	...	...	1	...	...	
Ascites .....	8	...	...	...	1	2	1	4	...	
Abdominal tumour .....	2	...	...	...	...	1	1	...	...	
Vomitus .....	1	...	...	...	...	1	...	...	...	
Cirrhosis .....	5	...	...	...	2	2	1	...	...	
Disease of rectum .....	2	...	...	...	...	...	2	...	...	
Dyspepsia .....	1	...	...	...	...	...	...	1	...	
Diphtheria and sore throat .....	5	...	1	3	...	1	...	...	...	
Stricture of pylorus .....	1	...	...	1	...	...	...	1	...	
Hæmorrhoids .....	1	...	...	...	...	...	...	1	...	
Disease of pancreas .....	1	...	...	...	1	...	...	...	...	
	114	...	4	15	14	35	27	18	1	
<b>CLASS VII.</b>										
Diabetes .....	16	...	...	2	1	6	7	...	...	
Renal .....	34	...	...	2	5	15	10	2	...	
Prostatic .....	10	...	...	...	...	...	4	3	3	
Cystorrhœa .....	1	...	...	...	...	...	...	...	1	
Disease of bladder .....	12	...	...	...	...	1	9	2	...	
Hæmaturia .....	4	...	...	...	1	1	1	1	...	
Supra-renal .....	2	...	...	1	...	...	...	1	...	
Cancer of kidney .....	1	...	...	...	1	...	...	...	...	
Disease of testicle .....	1	...	...	...	1	...	...	...	...	
	81	...	...	5	9	23	31	9	4	

TABLE I—continued.

Cause of Death.	Total.	Age at Death.								
		Und. 90	20 to 39	30 to 39	40 to 49	50 to 59	60 to 69	70 to 79	80 and above	
<b>CLASS VIII (see p. 281).</b>										
<b>CLASS IX.</b>										
Rheumatism .....	4	...	...	...	2	...	...	1	1	
Spinal .....	6	...	...	...	1	3	2	...	...	
Hip disease .....	1	...	...	...	...	1	...	...	...	
Lumbar abscess.....	1	...	...	1	...	...	...	...	...	
	12	...	...	1	3	4	2	1	1	
<b>CLASS X.</b>										
Carbuncle .....	7	...	...	...	...	4	2	1	...	
Anasarca .....	1	...	...	...	...	...	1	...	...	
	8	...	...	...	...	4	3	1	...	
<b>CLASS XI.</b>										
Suicide .....	7	...	...	...	4	2	1	...	...	
Sudden .....	3	...	...	...	...	2	1	...	...	
Accident .....	13	...	...	4	4	2	3	...	...	
Drowned .....	6	...	1	...	2	2	1	...	...	
Murdered .....	1	...	...	1	...	...	...	...	...	
	30	...	1	5	10	8	6	...	...	
<b>CLASS XII.</b>										
Old age and debility.....	43	...	...	...	...	1	8	19	15	
Unknown .....	13	...	1	2	3	4	3	...	...	
	56	...	1	2	3	5	11	19	15	

It is not intended to bring this forward as an unexceptional or final mode of arrangement; but in these and similar researches it seems to us that uniformity gives greater advantage than absolute perfection of method. All nosological arrangements are open to objection and liable to cross division of their contained classes; but it is a great and palpable benefit to be able at once to lay one beside another for comparison, exactly on the same footing. It is only a fresh application of the principle of percentage.

Class VIII is excluded from the general table, the female as well as the few lay deaths being given separately in tables of their own.

TABLE II.—*Deaths of Clerical Members.*

Class of disease.	Number dying.	Total years of ages at death.	Total years of ages at entry.	Duration of assurances.	AVERAGE—		
					Age at death.	Age at entry.	Duration.
1	88	4,221·0	3158·8	1067·7	50·8	38·0	12·8
2	60	8,566·1	2434·8	1131·3	59·4	40·6	18·8
3	245	14,254·6	9906·2	4349·4	58·2	40·4	17·8
4	185	10,144·6	7088·1	3056·5	54·8	38·3	16·5
5	149	9,178·7	6154·0	3024·7	61·6	41·3	20·3
6	114	6,384·9	4482·5	1902·4	56·0	39·3	16·7
7	81	4,869·0	3229·5	1639·5	60·1	39·8	20·3
9	12	682·1	510·3	171·8	56·8	42·5	14·3
10	8	477·1	332·1	145·0	59·6	41·5	18·1
11	30	1,481·2	1123·1	358·1	49·3	37·4	11·9
12—13	56	8,910·8	2514·4	1895·9	69·8	44·9	24·9
Total	1023	59,169·6	40,927·3	18,242·3			

TABLE III.—*Ages at Death in Decennial Periods.*

Class.	20 to 29.	30 to 39.	40 to 49.	50 to 59.	60 to 69.	70 to 79.	80 and upwards.	
1	5	15	16	27	17	8	...	
2	...	2	12	15	20	11	...	
3	1	21	43	65	71	40	4	
4	5	25	41	51	33	23	7	
5	...	6	14	41	51	33	4	
6	4	15	14	35	27	18	1	
7	...	5	9	23	31	9	4	
9	...	1	3	4	2	1	1	
10	...	...	...	4	3	1	...	
11	1	5	10	8	6	...	...	
12	1	2	3	5	11	19	15	
Deaths	17	97	165	278	272	158	86	= 1023
per 100	1·662	9·482	16·129	27·175	26·588	15·445	9·519	= 100

In the second table an abstract has been made of the twelve classes, excluding No. VIII, that of females, and the number of deaths, the duration of assurance, the total years of age at death as well as at entry have been collated in parallel columns. From these data, the average age at death in each class, the average age at entry, and the average duration of policies for the same lives have been computed. As average age at death is somewhat fallacious, where the cases are few and the differences great, a supplementary table of ages at death in each class divided into decennial periods has been added.

Lastly, the general totals have been collected, giving the whole number dying, the total duration of assurance, and the sum of years of life at death and at entry. The difference of the last two, of course, gives the previous figure.

TABLE IV.

Total years of age at death	.	.	.	59,169·6
"    "    entry	.	.	.	40,927·3
"    of duration	.	.	.	18,242·3

59,169·3 divided by number of lives (1028) = 57·8, average age at death.  
40,927·3   "      "      "      "      "      "      "      entry.

Average duration of policies	.	17·8
------------------------------	---	------

TABLE V.—*Lay.*

Cause of Death.	Total.	Age at Death.							
		Und. 90	90 to 99	80 to 89	40 to 49	50 to 59	60 to 69	70 to 79	80 and above
Carbuncle .....	3	...	...	2	...	1	...	...	...
Erysipelas .....	1	...	...	...	...	1	...	...	...
Pneumonia .....	3	...	...	1	1	1	...	...	...
Diabetes .....	2	...	...	...	...	...	...	2	...
Angina .....	1	...	...	...	1	...	...	...	...
Influenza .....	1	...	...	...	...	...	1	...	...
Phthisis .....	7	...	...	6	1	...	...	...	...
Renal .....	4	...	1	...	...	...	3	...	...
Bronchitis .....	2	...	...	...	1	...	1	...	...
Scarlatina .....	1	...	...	...	1	...	...	...	...
Morbus cordis .....	6	...	...	...	1	1	3	1	...
Accident .....	1	...	...	...	1	...	...	...	...
Enteric fever .....	1	...	1	...	...	...	...	...	...
Congestion of brain .....	1	...	1	...	...	...	...	...	...
Cancer .....	3	...	...	...	3	...	...	...	...
Hepatic .....	4	...	...	...	1	1	2	...	...
Hæmaturia .....	2	...	...	...	1	...	1	...	...
Abcess of knee .....	1	...	...	...	...	...	1	...	...
Hæmoptysis .....	1	...	...	...	...	...	1	...	...
Asthma .....	1	...	...	...	...	...	1	...	...
Decay .....	1	...	...	...	...	...	...	1	...
Aneurism .....	1	...	...	...	...	...	1	...	...
Apoplexy .....	1	...	...	...	...	...	1	...	...
Cerebral .....	3	...	1	...	1	1	...	...	...
Pyæmia .....	1	...	...	...	1	...	...	...	...
Unknown .....	1	...	...	...	1	...	...	...	...
Delirium tremens .....	1	...	...	1	...	...	...	...	...
	55	...	4	10	15	8	16	2	...

TABLE VI.—*Laumens.*

Total years of age at death	.	.	.	2742.7
"    "    "    entry	.	.	.	2013.0
"    "    duration	.	.	.	729.7

2742.7 divided by number of lives (55) = 49.9 average age at death.  
2018-0 " " " = 86.6 " " entry.

Average duration of policies . 18.3

TABLE VII.—*Females.*

Cause of Death.	Total.	Age at Death.							
		Und. 20	20 to 29	30 to 39	40 to 49	50 to 59	60 to 69	70 to 79	80 and above
Apoplexy .....	5	...	...	...	...	3	...	2	...
Paralysis .....	3	...	...	1	...	...	1	1	...
Phthisis .....	7	...	...	2	3	2	...	...	...
Hepatic .....	4	...	...	...	1	...	3	...	...
Pneumonia .....	3	...	...	...	1	1	1	...	...
Morbus cordis .....	2	...	...	...	...	...	...	1	1
Fatty heart .....	1	...	...	...	1	...	...	...	...
Cancer .....	5	...	...	1	...	1	2	1	...
Accident .....	1	...	...	...	1	...	...	...	...
Old age .....	3	...	...	...	...	...	...	2	1
Bronchitis .....	2	...	...	...	1	...	...	1	...
Albuminuria .....	1	...	...	...	...	...	...	1	...
Paraplegia .....	1	...	...	...	...	...	...	1	...
Climacteric .....	1	...	...	...	...	...	...	1	...
Uterine .....	1	...	...	...	...	1	...	...	...
Cystitis .....	1	...	...	...	...	...	...	1	...
Spinal .....	2	...	...	...	...	...	1	1	...
Unknown .....	2	...	...	...	...	...	2	...	...
Ascites .....	1	...	...	...	...	...	...	1	...
Puerperal .....	3	...	1	1	1	...	...	...	...
Influenza .....	1	...	...	...	...	...	...	1	...
Fever .....	1	...	...	1	...	...	...	...	...
Ovarian .....	1	...	...	...	...	1	...	...	...
Disease of bowels .....	1	...	...	...	1	...	...	...	...
Diarrhoea .....	1	...	...	...	...	...	...	1	...
Peritonitis .....	1	...	...	...	...	...	1	...	...
Erysipelas .....	1	...	...	1	...	...	...	1	...
Suicide .....	1	...	...	...	...	...	...	1	...
	57	...	1	7	10	13	14	10	2

TABLE VIII.—*Females.*

Total years of age at death	.	.	.	.	.	.	8254·5
" " " entry	.	.	.	.	.	.	2554·9
" " duration	.	.	.	.	.	.	699·6
3254·5 divided by number of lives (57) = 57·1 average age at death.							
2554·9 " " " = 44·8 " " entry.							
Average duration of policies	.	12·5					

The first point requiring notice is that the clerical lives were insured for an average period of 17.8 years, having come in at the average age of 40 and died at the average age of 57.8.

The laymen seem to have entered at a somewhat earlier age, viz. 36.6, and to have died considerably sooner, viz. at 49.9, giving an average duration for their insurances of 13.3 years.

The females entering at an average of 44.8, i. e. more than four and a half years older than the clergymen, and eight years older than the laymen, died at an average age of 57.1, giving only a duration of 12.5 years.

This result singularly corroborates the general impression existing in this office, that female lives have not paid so well as male. Nor can the difference be due to puerperal causes alone, for the average age of entry is highest in the female class, viz. 44.8, a time at which the specific risks of female existence may be reasonably supposed to have all but ceased. No doubt in so restricted an experience a few unlucky instances may have had serious effect on the result, and gone far to justify the unfavorable estimate named above. On the other hand, only three cases in all are set down to puerperal disease and a single case to peritonitis.

In the above tables two points generally deserve notice; first, the most prominent causes of death whether in classes or as single diseases, and, secondly, common diseases, the absence or infrequency of which in these statistics is remarkable.

As regards the former the class of cerebral diseases leads with 245 deaths; although the average duration of the lives in that class, viz. 58.2, is somewhat above the general average for all the lives, viz. 57.8.

Respiratory diseases follow at rather a long interval with 185, the average length of life being 54.8, or three years less than the general average. Circulatory disorders give 149, with a life average of 61.6, a figure only exceeded by the admittedly senile deaths. Digestive diseases come fourth, with 114 deaths and an average life of 56 years. Epidemic diseases sink to a remarkably moderate number of 83; although, for obvious reasons, the duration of life is only 58 years. Urinary disorders fall still lower, giving only 81 deaths and a high average life of 60.1 years.

The principal diseases to which deaths have been due require and deserve separate tabulation.

TABLE IX.—*Principal Causes of Death.*

Class.	Disease.	No. of deaths.	Average.		
			Duration.	Age at death.	Age at entry.
2	Cancer .....	87	17·1	55·7	38·6
3	Apoplexy .....	92	18·8	58·9	40·1
	Paralysis .....	65	19·7	62·7	42·9
	Cerebral .....	64	16·2	55·7	39·5
4	Bronchitis .....	48	25·0	66·9	41·9
	Pneumonia .....	88	18·6	56·0	39·4
	Consumption .....	62	10·9	45·1	34·2
5	Heart diseases .....	110	20·5	62·4	41·9
	Angina .....	16	20·1	62·2	42·1
6	Liver diseases .....	27	15·0	52·5	37·5
7	Diabetes .....	16	19·9	57·1	37·2
	Renal diseases .....	34	18·8	56·5	37·7
12	Old age, &c.....	43	28·4	76·4	48·0

It will here be seen that heart disease stands at the head of the list, with 110 deaths. Apoplexy follows with 92, and paralysis with 65. If the two latter be combined, as they probably ought to be, the figure rises to 157, and thus over-tops the first named.

Grouping together the prominent brain affections, and extending the basis to all the lives, clerical, lay, or female, the deaths in Class III rise to 234 (or 20·6 per cent.) as against 172 from bronchitis, pneumonia, and consumption, and 119 from diseased heart. The great predominance of nervous affections in advanced life is here brought out prominently, and the opinion that they may be looked on as almost a natural termination of old age is corroborated. There are, however, 43 entries under the simple heading of "old age and debility," 19 of which were between the age of 70 and 79, and 15 were 80 years old and upwards. These are, when separated from the "unknown" deaths, Class XII gives the high average age at death of 76·4 years and 28·4 of duration of insurance. In connection with this topic it seems desirable to continue up to the end of the last financial year the history of the first 90 policies issued by the Society which was carried by us in our previous

paper up to the date of our investigation (viz. 30th November, 1872). The summary then given was as follows:

*Summary.*

	Number of policies.	Sum assured.	Additions after total extinction of premium.	Original annual premium.	Premium payable as reduced by bonus.	Average age at entry.	Average age at exit or at date.	Average duration.
Claims by death	65	£ 38800	£ 5657	£ 1211 16 11	£ 309 18 11	Years. 38½	Years. 66	Years. 27½
Surrenders .	6	3100	158	94 15 8	61 1 5	86	44	8
Arrears .	5	2150	...	61 2 1	61 2 1	32	34	2
Existing 30th Nov., 1872 .	14	8900	4542	218 9 8	Nil.	Years. 33½	Years. 76	Years. 42½
	90	52350	10357	1586 4 4	432 2 5	—	—	—

Since the date of these observations 3 policies only have become claims, at an average age of rather more than 82 years, giving a duration of over 41 years for each policy. Adding these 3 claims to the 65 given in the above table, the average age at entry remains unchanged, viz. 38½ years, the average age at death is increased by  $\frac{1}{2}$  a year, viz. from 66 to 66½ years, and consequently the average duration from 27½ to 28 years.

The particulars of the 11 policies existing on 31st May, 1876, are subjoined, and also a summary of the 90 policies to the same date. These tables clearly indicate that the assured members have just cause for congratulation in the judicious selection of lives made by the founders.

*Policies existing on 31st May, 1876, out of the first 90 issued by the Society.*

Policy No.	Sum assured.	Additions after total extinction of premium.	Age.		Original annual premium.	Premium now payable.	Number of bonuses allotted.
			Entry.	At date.			
21	500	663	40 1	86 3	15 10 10	Nil.	9
24	200	160	83 2	79 4	5 0 0	„	9
26	600	280	26 0	72 2	12 8 0	„	9
108	1000	1144	87 2	83 3	29 3 4	„	9
142	1000	399	23 11	69 11	19 10 0	„	9
264	500	365	32 3	77 9	12 10 0	„	9
291	1000	920	35 2	80 6	27 6 8	„	9
330	800	631	38 9	78 10	20 12 0	„	9
331	100	120	39 11	85 0	3 2 2	„	9
362	100	40	26 8	71 6	2 2 6	„	9
364	500	359	33 0	77 9	12 17 6	„	9
(11)	6300	5081	361 1	862 3	160 3 0	Nil.	

*Summary as at May 31st, 1876.*

	Number of policies.	Sum assured.	Additions after total extinction of premium.	Original annual premium.	Premium payable as reduced by bonus.	Average age at entry.	Average age at exit or at date.	Average duration.
Claims by death	68	40800	7065	1270 3 7	809 18 11	38½	66½	28
Surrenders	6	3100	158	94 15 8	61 1 5	36	44	8
Arrears	5	2150	...	61 2 1	61 2 1	32	34	2
Existing 31st May, 1876	11	6300	5081	160 3 0	Nil.	33	78½	45½
	90	52350	12304	1586 4 4	432 2 5	—	—	—

There remain, for consideration a few of the diseases which are remarkable by their infrequency in the course of these statistics, as compared with the general mortality of unselected lives. Foremost among these stand, delirium tremens and hernia, which during 45 years have only contributed a single death each. Next stand rheumatism and insanity, with only four deaths, gout with five.

Renal diseases, although furnishing the higher figure of 39 deaths, are singularly below their proportion as found in ordinary mortality tables, and the same is true in a higher degree of consumption, which only contributes 76 cases, on a total considerably over a thousand, or about 6 per cent. of all deaths. It is obvious that careful medical selection has materially affected this item.

Taking the three principal diseases of the thoracic organs, namely, bronchitis, pneumonia, and consumption, we have only 172 deaths against 234 from apoplexy, paralysis, and cerebral disease.

The last table is a comparison of experience from eight of the most important offices, reduced to a uniform scale for greater facility of reference.

TABLE X.—*Causes of Death—Comparative Experience of Life Offices.*

Causes of Death.	Proportional number of deaths in each class per 1000 deaths.							
	Clergy Mutual. Deaths 1023.	Metropolitan. Deaths 871.	Scottish Widow. Deaths 3307.	Scottish Amicable. Deaths 778.	Standard. Deaths 1516.	London Life. Deaths 3894.	Eagle. Deaths 3090.	North British. Deaths 1308.
1. Epidemic, &c.	81	95	143	194	133	110	142	143
2. Uncertain seat.	59	76	66	89	45	55	101	106
3. Brain, &c.	239	224	223	168	197	240	185	198
4. Respiratory	180	244	207	239	237	201	212	211
5. Heart, &c.	146	107	111	87	98	117	63	94
6. Digestive organs	112	121	126	125	143	122	95	104
7. Urinary organs	79	52	46	80	44	39	18	32
8. Uterus and childbirth	Nil.	2	5	4	3	4	19	4
9. Joints and bones	12	6	9	9	5	4	4	2
10. Integumentary	8	6	1	5	2	3	6	2
11. Violence and accidents	29	30	28	56	47	26	24	85
12. Decay and old age	65	87	24	21	29	62	74	88
13. Causes unknown	—	—	13	28	17	17	57	31
	1000	1000	1000	1000	1000	1000	1000	1000

## NOTE

IN REFERENCE TO THE

# WELSH LL AND CERTAIN OTHER SURD OR ASPIRATE CONSONANTS.

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By J. S. BRISTOWE, M.D., F.R.C.P.

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In the first volume of our Reports, issued in 1870, I published a paper on the "Mechanism of Articulate Speech," which I desire to correct and extend.

In the first place, by an unfortunate error, which I confess with some degree of shame, I employed the terms sonant and surd in reference to letters in exactly the contrary sense to that in which they were primarily and are properly employed; and hence I should wish any one who cares to peruse that paper to transpose these terms wherever he meets with them there. The error arose from a grammatical slip in the first edition of Latham's 'English Language,' from which book I first, many years ago, learnt something of the mutual relations of letters. He mentions the terms once only, and then as synonyms of other terms having a similar application; and in indicating their meaning refers to them in such a way that the term sonant is made to appear synonymous with sharp, and the term surd synonymous with flat.

I easily satisfied myself that the term sonant was properly applied to the sharp or higher tone of such letters as p, t, k, f, s, and the like, and that the term surd was properly applied to the deeper and less penetrating sounds of b, d, g (hard), v, z, and such like; and as the expressions are not very commonly used I failed, until quite recently, to correct my error. I need

scarcely now add that the sonant letters are really those which are attended with laryngeal intonation, and the surd those which are effected solely in the oro-nasal cavities.

My main object, however, in writing this addendum to my original paper is to extend the tabular scheme of consonants which is there given. For I did not at that time fully appreciate certain consonantal sounds which I now know to be distinct elements of articulate utterance.

*The first of these is the Welsh consonant represented by Ll.* This I had believed, with most Englishmen, to be a compound sound formed of th (in thing) followed by an ordinary l; and that the word "Llangollen," for example, was properly represented in English spelling by the following combination of letters, "Thlangothlen." But about eighteen months ago, when in Towyn, North Wales, I made the acquaintance of Dr. Charles, a distinguished Welsh scholar, and discussed with him the nature of the sound represented by the double l. He did not pretend to explain the nature of the sound, or to say whether he regarded it as a single consonantal sound like s or t, or (if one may be allowed to use the expression) as a diphthongal consonantal sound, such as that represented by x (in Essex), j (in June), or ch (in Charles); but he pronounced it for me over and over again, both without vowel assistance and in various combinations. I then clearly recognised that *thl* was not the English equivalent of *ll*; but was inclined to think the true sound of *ll* was represented by that of the German *ch* (in Ich) followed by *l*, and consequently that Llangollen might be spelt *Chlangochlen*. After a while, however, I learnt to articulate the sound of *ll* accurately, the accuracy of my pronunciation being confirmed by Dr. Charles; and I then satisfied myself beyond all doubt that the double *l* is really an elementary articulate sound, not existing in the English language, or, so far as I know, in any other than Welsh, and that it is, in fact, the surd equivalent of the ordinary sonant *l*, that is to say, it holds exactly that relation to the *l* of English, German, and French, that *f* holds to *v*, that *th* (in thing) holds to *th* (in the), that *s* holds to *z*, and that *s* (*sh*) (in Asia) holds to *z* (*zh*) in azure. It is the sound of *l*, as evolved by the rush of air past either side of the tongue, unattended with laryngeal intonation or even laryngeal sound of any kind.

When writing my former paper I failed to recognise the fact that surd varieties existed of any sonant letters, excepting the explodents and the sibilants; and accordingly in the scheme of consonants which accompanied that paper I made a place for such varieties in these two cases only. My newly acquired knowledge, however, of the meaning of the symbol *ll* has made me examine with much more care than I formerly did the question of the actual existence of surd and sonant varieties of other letters. The following are the results at which I have arrived.

*The surd w.*—Formerly I was inclined to regard the aspirated *w* in “what” and “which,” as pronounced more particularly by the Irish and Welsh, as simply an ordinary sonant *w* preceded by an ordinary aspirate; that the aspirate has, indeed, the same relation to the consonant in this case that it has to the vowel in such words as “he” and “hoop.” I am satisfied, however, now that the *wh* in these words and words like them, when it has a sound different from that of the ordinary *w*, is not represented phonetically by *hw*, that it is not a double sound, and that there is no real aspirate, but that it is the surd form of the ordinary sonant *w*.

*The surd r.*—The true *r*’s are formed with the aid of the tip of the tongue. Of these there are, as I endeavoured to show in my previous paper, two forms: one trilled, the other untrilled and, according to my view, a semivowel belonging to the same series as *y* and *w*. Both of these distinctly present surd varieties. But neither of these surd sounds belongs to the English language. In the endeavour to aspirate the *r* (a performance which is easy of accomplishment) persons generally, I believe, enunciate its surd variety in place of aspirating the sonant letter; they utter a sound which is in no sense a true aspirate, nor a compound of the aspirate and the sonant *r*, but an independent letter having the same relation to the ordinary *r* that *ll* has to *l*. I think it not improbable that the Greek aspirated *r*, *ρ*, was really this letter.

*The surd y.*—This sound also exists as a modification of the ordinary *y*. Theoretically it should, I think, be regarded as distinct from the sound of *ch* in the German *Ich*. But I confess that I cannot with my own organs of articulation make any difference between them which is appreciable to my ear.

*The surd nasal continuants, ng, n, and m.*—These, again, can undoubtedly be enunciated. But I must acknowledge that in endeavouring to pronounce them they seem to me scarcely distinguishable from one another, the distinctive sounds, feeble from the beginning, losing themselves in the common sibilant sound due to the rapid passage of air through the nasal passages. I am by no means clear, however, that with some little education the ear might not be able to distinguish readily the shades of difference between them.

In order to explain the meaning of the accompanying table of consonants, I will quote, with slight modification, a paragraph from my original paper.

It will be seen that in it I have arranged the consonants in three vertical groups—the labial, the lingual, and the guttural—and have placed beneath each of these groups the vowel which seems to me to have a special affinity with it. Each of these groups comprises nasal, explosive, sibilant, trilled, and semivowel consonants, which, again, are subdivided into surd and sonant modifications. It will be observed that in the guttural and labial groups I have made two parallel vertical series, and in the lingual group three such series, and that I have represented some of the letters by small characters, some by capitals. The object of this is partly to show that the same letters, somewhat modified in character, may be produced by somewhat modified arrangements of the vocal organs, but chiefly to show that certain well-characterised letters fall naturally into series, of which the other members comprise these modified consonantal sounds only. In all cases the capital letters indicate the position in the scheme of what I may call the perfect letters, at any rate, of those letters which seem perfect to English ears and English organs of speech; the small letters give the position of the consonants which are a modification of these. The small letter *h* has been placed after those surd modifications of sonant letters which have no recognised characters of their own, simply because the addition of *h* to *w* and *r* is the usual mode of indicating the surd or so-called “aspirate” varieties of these latter letters.

Those who wish to have a thorough understanding of the table are referred to my original paper in the first volume of the Reports, p. 119.

*Scheme of Consonants.*

		GUTTURAL.		LINGUAL.		LABIAL.			
		Base of tongue and uvula.		Base of tongue and palate.		Dorsum of tongue and palate.		Tip of tongue and palate.	
Nasal continuants	NG	n		N		n		m	M
	NGh	?		Ng		?		?	Mh
Explodents	G	d		D		d		b	B
	K	t		T		t		p	P
Sibilants.	gh	ZH		Z		DH		v	V
	?	Kh		SH		TH		f	F
Oral continuants	l	L		l		l		sonant	
	?	Ll (Welsh)		?				surd	
Trills.	r	R		R				r	Sonant
	p	P						?	Surd
Semicivowels	Y			R				W	Sonant
	Yh			Rh				Wh	Surd
Vowels of each group of consonants		E						OO	
“Ur vocal”									



## THREE CASES

OF

## IMPERFORATE RECTUM.

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By H. H. CLUTTON, F.R.C.S.,  
RESIDENT ASSISTANT SURGEON.

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THROUGH the kindness of Mr. Mac Cormac three cases of imperforate rectum have come under my care during the last six months. As one of them has been successful, and another, although less fortunate, not less instructive, I have ventured to bring forwards the notes of these cases for the 'St. Thomas's Hospital Reports.'

On September 7th, 1876, W. T. E—, a healthy well-nourished male child, of full time, and scarce 48 hours old, was admitted into St. Thomas's Hospital. He had passed nothing per anum. Abdomen distended. Constant vomiting.

On examination a perfectly formed anus was found, but it would not admit more than the tip of the little finger. The anal *cul-de-sac* was, therefore, enlarged by an incision backwards towards the coccyx, so as to more thoroughly explore the contents of the pelvis. On introducing the finger a tumour could be felt with a distinct impulse as the child cried. This, however, seemed placed so far forwards as to make it doubtful whether it were the bladder or the distended rectum. But a cord-like structure could be felt passing from the anal *cul-de-*

*sac* towards the presumable rectal termination of the intestine. A catheter also passed into the bladder had no influence in diminishing the size of this tumour.

Mr. Wagstaffe, who was present at the operation, also examined the case, and recommended the exploration with a trocar. A small trocar was, therefore, introduced into the centre of this tumour, with the result of a copious evacuation of meconium through the canula. As the fluid seemed to escape freely the canula was tied in. The child was very much relieved the next day, had no more sickness, and took to the bottle well. The canula was removed forty-eight hours after the operation, having attained its object of relieving the obstruction, and a piece of laminaria tent was introduced and left in the wound for a few hours. Upon its removal at first a No. 16 catheter was passed every day, and then a rectal bougie, gradually increasing the size from No. 1 to No. 4. And at this time the little finger passed easily into the bowel without any constriction. The object here was to produce a gradual dilatation of the communication between the anus and the rectum, for since the anus itself was naturally formed, and the tissue between the two closed *sacs* was extremely thin, it was not thought expedient to expose the child to the additional risk of dissection in order to bring the bowel down to the margin of the anus. To do so in such a case would be to make one mucous membrane overlap another.

The child did exceedingly well for three weeks and then began to fade. He grew thinner, refused the bottle, and at last was covered with profuse eruptions of red-gum. At the same time numerous small abscesses formed in different parts of his body. He was given grey powder every other night, cow's milk was substituted for condensed, and cod-liver oil rubbed into his skin wherever it was free from inflammation.

He gradually picked up under this treatment and regained his former strength. But it was noticed that he had during the latter part of this time been straining and crying a great deal whenever he passed a motion, and on examining the rectum it was found there was a tight constriction at the seat of the former operation. The finger and bougie treatment which had been omitted during the child's illness was resumed. This was continued from November 18th till January 22nd,

when the following note was taken :—" Not the slightest dilatation has been effected, and the constriction is so tight as to be positively painful to the finger, which is arrested at the first phalangeal joint."

A hernia knife was therefore passed through the stricture, and the division made in three places towards the sacrum. The bougie or finger was passed after this regularly every day, but notwithstanding this persevering treatment the stricture again contracted and required division on March the 7th. The thickness of the surrounding tissue certainly diminished considerably after the first division.

That the child has recovered is, indeed, satisfactory, considering the high mortality that attends this congenital malformation. But the question arises, ought the resulting stricture to have been avoided? Ought the additional risk to have been incurred to avoid a subsequent deformity? Where there is no natural anus it seems only rational to pull down the mucous membrane and attach it to the margins of the incision, and this is comparatively easy where the gut is not far removed from the surface. But if the rectum be high up in the pelvis there is an increased risk incurred in bringing the bowel down, for it would require some dissection and force to detach it, and sometimes, indeed, it is practically impossible if you have any respect for the tissues of the pelvis, as the following case will, I think, show.

G. A—, male child, 2 days old, was brought to St. Thomas's Hospital on February 22nd, 1877. The anus was found in its normal position, terminating in an exceedingly dense tissue about half an inch from the cutaneous margin. No impulse could be felt. The bladder was emptied by the catheter, and a silver probe introduced to act as a guide to the position of the bladder during dissection. The sphincter ani was slit up backwards and dissection carried on along the anterior surface of sacrum. Although the dissection was suspended every now and then and impulse carefully sought for whilst the child was crying, not the slightest indication of the position of the bowel could be obtained. In a few minutes, however, a drop of meconium could be seen by the side of the knife. A director was passed by the side of the

knife into the rectum and the knife withdrawn. The discharge was immediately profuse. The opening was then enlarged towards the sacrum by passing the knife along the groove of the director. On introducing the finger the aperture in the rectum could be felt, as far as one could judge, rather more than two and a half inches from the margin of the anus, and immediately in contact with the sacrum in the central line, to which it seemed firmly and closely connected. The tip of the index finger just entered the aperture in the rectum when buried beyond the second phalangeal joint. This child, although exceedingly healthy looking and well nourished, died three days afterwards from convulsions.

In this case it was practically impossible to bring the bowel down with the forceps, for in the first place it was so high up that one would have been fishing about in the dark and, no doubt, have brought down something, but one could hardly say whether it would be the bowel or not before it appeared. If in these cases the incision were enlarged so as to throw some light into the wound, with careful sponging the mucous membrane might be seen, or forceps might be introduced with the finger and the margin of the gut felt for, but without the assistance which the increased size of wound would give I do not see how it can be done. And is it right to run this additional risk which such an enlargement of the wound would necessarily entail upon the patient? Mr. Le Gros Clark had an exceedingly interesting case which I think may throw some light upon this point. It is referred to both by Mr. Curling 'On Diseases of the Rectum,' and by Mr. Holmes 'On Diseases of Children.' In the latter is the following passage referring to Mr. Clark's case:—"The anal *cul-de-sac* was about half an inch deep, and the obstructing tissues were estimated by Mr. Clark to have been as much as two inches in thickness. The operation was performed with a straight narrow bistoury, with which the tissues were freely divided in the middle line, the incisions being carried from before backwards. No measures were taken at first to obviate the contraction of the parts, so that the passage recontracted and constipation and vomiting set in. This was due to the formation of a cicatricial tissue, which required division with a hernia knife and tearing open with dressing forceps, followed

by the daily use of bougies. Under this treatment the contracting tissues lost their hardness and resistance, and at the date of Mr. Clark's paper the surface of the track was beginning to feel soft as if something like a mucous membrane were being formed upon it." And Mr. Curling adds, "and about ten months after the second operation he (Mr. Clark) again divided an obstructing band with a hernia knife. Dilatation was afterwards persevered in. The boy at nine years of age was in tolerable though not robust health, but subject at times to serious troubles in defecation."

Mr. Le Gros Clark has himself told me quite recently that this patient is now a married man about twenty-five years of age, that he has little trouble now, and nothing more than what he himself can manage without the assistance of a surgeon.

Such is the sequel of a case where the gut was not pulled down, and resulting contraction followed. Yet in the end this has been a complete success.

Mr. Curling describes a case where there was an anal *cul-de-sac* in which he drew down the bowel and secured it to the wound in the skin, but the depth at which the bowel was seated was not more than an inch. In this case the subsequent contraction of the passage has been entirely obviated. At four years of age he "was a remarkably fine boy, with the passage quite free."

There can, I think, be no doubt that where the gut is within easy reach it should be done even if there be an anal *cul-de-sac*, but it should not, I think, be persevered in as a matter of vital importance. Babies of that age are little able to withstand the effects of a cutting operation, and anything added thereto, such as dragging upon the tissues of the pelvis, cannot but be detrimental. Is it possible that the high mortality in these cases is in some measure due to operative interference, however necessary it may be? It is curious to note, and I think it is as yet unexplained, how frequently they gradually fade away and die in a marasmic condition in a fortnight or three weeks, although they seem to recover from the direct effects of the operation.

On January 24th, 1877, a male child, 4 days old, was

brought to St. Thomas's Hospital, said to be without a lower aperture to its bowel.

On examination the anus was found in its normal position, but terminated in a *cul-de-sac* at about half an inch from the surface.

Mr. Mac Cormac, who was present, and under whose care it would have in due course come, very kindly gave the treatment of the case into my hands.

The anal *cul-de-sac* was enlarged by cutting backwards towards the coccyx, and although no impulse could be felt when the child cried, it was thought advisable to carry the dissection into the pelvis. After dividing the tough tissue which formed the apex of the anal aperture, the finger easily entered the cavity of the pelvis, and without having much recourse to the use of the knife, and carefully following the concavity of the sacrum, the promontory was eventually reached. During this dissection, with the most careful examination not the slightest impulse was felt, although the child was crying forcibly. It was evidently imprudent to carry the dissection higher. A probe was therefore passed into the bladder with the object of more thoroughly exploring the tissues of the pelvis. As the probe passed down the urethra with the finger in the wound they were in such close contact that it seemed impossible the dissection could have been carried more forwards without wounding the urinary organs, and as the finger was placed upon the sacrum the faintest hope of there being any gut in the cavity of the pelvis was given up. Mr. Mac Cormac also examined the case in the same way and came to similar conclusions.

The risks of any further operative measures were put before the child's friends, as well as the inevitable result if left to itself, but they declined sanctioning the only alternative until they had consulted the infant's mother.

The child was brought back on the following day (5th) by the friends, and we were requested to do what we could to save life.

Abdomen still more distended, but no sickness. From the doctor's orders no milk or food had been given to him. After the thorough search that had been made on the previous day

for the rectum in the pelvis it was not thought advisable to reopen the wound.

Littré's operation in the left inguinal region was therefore performed, the gut being stitched to the margin of the wound before opening it.

26th.—Profuse discharge of meconium throughout last night, and during last few hours yellow faeces. Abdomen less distended and quite flaccid to touch. Takes milk well from the bottle.

27th.—Going on well. Slightly constipated. Tendency to sickness.

29th.—Bowels have acted thoroughly. Sickness has stopped. No tenderness of abdomen. Has the thrush very badly.

30th.—All stitches taken out. Poultice applied to wound, which looks rather inflamed. No sickness. No tenderness of abdomen.

Feb. 3rd.—Child does not seem to pick up at all, but rather gets thinner and thinner every day. He had no signs of peritonitis.

From this date the child gradually faded away; its skin at the last hung in folds from the extremities, and reduced to the most extreme degree of emaciation, he died on February 12th, eighteen days after the operation and on the twenty-first day of his life.

*Post-mortem.*—The artificial anus in the left groin communicated with sigmoid flexure. No disease in any organ. No trace of peritonitis. The large intestine occupied the normal position in the abdomen.

The rectum was found in the pelvis containing faeces, displaced slightly to the left, but passing down to within an inch and a half of natural anus. There was no trace of any fibrous cord uniting the anal *cul-de-sac* with the termination of the rectum. This had, no doubt, been divided in the first operation.

How the rectum escaped being discovered and opened in the dissection that was carried high up in the pelvis it is difficult to say. Perhaps from too great a fear of injuring the bladder the dissection was carried too far back towards the sacrum, and the left forefinger used in pushing aside the structure where the knife would have been more applicable, and thus the rectum

dissected from off the sacrum. But undoubtedly the rectum must have been empty at the time of the first operation or it could not have been missed when feeling for it with the probe in the bladder. This brings us to another point, and that is that the pelvis ought to have been explored a second time by reopening the wound before proceeding to colotomy, for in the interval of a day the meconium might have descended lower and formed a tumour easily recognisable in the pelvis. But in the case G— exactly the same method of operating was adopted; the left forefinger in the wound carefully indicated the exact spot that it was desirable to incise, so that step by step the tissues in front of the sacrum were gradually and thoroughly explored. In the one case the rectum was firmly adherent to the sacrum, and so was readily opened, and this close attachment one could subsequently feel and appreciate, and in the other case the rectum must have had a more or less greater length of meso-rectum and so was easily pushed aside.

It might, perhaps, be said by some surgeons that if a trocar had been used and thrust in a direction parallel with the sacrum the rectum would not have been missed, but this does not agree at all with the history of the operations for this mal-formation. There are numerous cases on record in which the trocar has been used and has failed to find the gut although present in the pelvis. In one case a fluctuating tumour was in this way punctured, which subsequently proved to be the recto-vesical pouch, and the trocar had passed by the side of the rectum. By dissecting carefully with the knife along the sacrum the bowel would probably have been reached before the pouch. The trocar can only explore a limited portion of the pelvic cavity, and where there is no tumour to be felt the thrusting of a trocar, one might almost say at hap-hazard, must be attended with considerable risk; of course the condition that calls for this operation is one that admits of a certain amount of danger being incurred, but the finger acting as a guide to the knife must be a safer and surer method than the trocar; and if the method of examining the pelvis by passing a probe into the bladder were more frequently resorted to during the dissection the exact condition of things would be more often discovered.

In discussing the question as to whether colotomy should be

performed in these cases an argument might be derived from this case. And there are other instances on record in which the rectum has been similarly missed and colotomy been performed. Here a probe might subsequently have been passed down from opening in the groin, and made to present in natural position of anus (as was, indeed, contemplated had the child lived), and the deformity thus remedied.

Velpeau, I believe, was the first to make this suggestion, after having performed colotomy, and at the post-mortem examination discovered the rectum in the pelvis empty and deviating to the right. But it does not seem that this suggestion has ever been carried out.



REPORT OF  
ST. THOMAS'S HOSPITAL MEDICAL AND  
PHYSICAL SOCIETY.

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*Officers.*

*President and Treasurer.*—DR. TURNER.

*Vice-Presidents.*—THE PHYSICIANS, SURGEONS, ASSISTANT PHYSICIANS, ASSISTANT SURGEONS, A. O. MACKELLAR, ESQ., AND LECTURERS.

*Hon. Secs.*—MR. JACOB, MR. ROSSITER.

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REPORT OF PROCEEDINGS, 1875-6.

*October 14th, 1875.*

The PRESIDENT gave an inaugural address. He noticed the size and usefulness which the Society had attained under the able Presidentship of Mr. Wagstaffe. He advocated the introduction of discussions independently of ready written papers, and reviewed the advantages to be gained by members from such a custom. The President also suggested the treatment of points of difficulty encountered by students, as thereby not only giving the means by which such subjects might be well investigated, but also that others should become the partakers of the result of such good work. The address furnished material for the first meeting.

*November 4th.*—Dr. CORY in the Chair.

Mr. BARKER read a paper on "Diet." He spoke of the conditions which regulated demand on individual requirements and on the systems of dieting large bodies of men.

Members of Committee for the first year were elected.

*November 25th.*—The PRESIDENT in the Chair.

Mr. DAVIES read a paper on "Consanguineous Marriages." His proposition was, that "Marriages of consanguinity are dangerous, not as, *per se*, tending to produce degeneracy of race, but by the tendency they have to strengthen and develop in the offspring certain taints or idiosyncrasies peculiar to father or mother or both."

*December 9th.*—The PRESIDENT in the Chair.

Mr. LONGSTAFF read a paper on "Digestion in Plants." He specially alluded to the phenomena of the insectivorous plant *Drosera rotundifolia*, described the nature and action of its secretion, and the influence of drugs upon it. The organic food assimilated by this and similar plants, said Mr. Longstaff, served to break down another of those distinctions drawn between the animal and vegetable kingdoms.

*December 22nd.*

The Annual Microscopical Soirée was held in the library, when the following and other specimens were exhibited :

TABLE I. Dr. TURNER. Miscellaneous.

- „ 2. Dr. REID. Microscopical Sections (various).
- „ 3. MESSRS. MILLIKIN. Surgical Instruments, &c.
- „ 4. Mr. RAINES. Sections of Thymus Gland, &c.
- „ 5. Dr. GREENFIELD. Diseased Spinal Cord.
- „ 6. Dr. ORD. Blood-crystals and Cell Formation.
- „ 7. Mr. STEWART. Miscellaneous—Polariscope.
- „ 8. Statuary—lent by Mr. OSBORNE.
- „ 9. Mr. WAGSTAFFE. Tumours.
- „ 10. Mr. BERNAYS. Ophthalmoscope, &c.
- „ 11. Dr. HARLEY. Parasitism of the Mistletoe.

**TABLE 12. Messrs. MURRAY and HEATH. Views and Miscellaneous Microscopic Objects.**

„ 13. Mr. SYDNEY JONES.

„ 14. Mr. C. STEWART. Microscopical Specimens of Food.

In the Physical Room Mr. JACOB demonstrated—Phosphorescence, Fluorescence, Absorption Spectra, &c.—by instruments kindly lent by Dr. STONE.

Specimens of Haematite and Iron Manufacture—kindly lent by Dr. BERNAYS.

*January 13th.—Dr. ORD in the Chair.*

Mr. FREUND read a paper on “Diarrhoea.” He prefaced his remarks on the classification of the forms of diarrhoea by a résumé of the physiology of the bowel.

*January 26th.—Mr. STEWART in the Chair.*

Mr. GIMLETTE read a paper on “Antiseptic Surgery.” He advocated antiseptic treatment in all cases where suppuration might be dangerous; believing that, as putrefaction and suppuration are indeed caused by the introduction of germs from without, so their exclusion will prevent these processes. The reader severally noticed the antiseptic agents in common use.

*February 17th.—The PRESIDENT in the Chair.*

Mr. BERNAYS read a paper on the “Treatment of Inflammation.” Regarding the dilated vessels and open mouths of lymphatics as the most obvious channel for escape of inflammatory products, and stating the retention of these products to be the cause of the most disastrous results in inflammation, the reader argued that the application of cold to inflamed parts could only be justified when these products could be removed by other means, as, for instance, in mucous membranes. And since the contraction of vessels is not desirable in acute inflammation, it is therefore important to remove blood, when required, by means which do not contract the blood-vessels, as is done in the use of wet cups.

*March 9th.—The PRESIDENT in the Chair.*

Mr. BALLANCE read a paper on "Life." He criticised the various definitions of life given by celebrated men, considering them useless, as being either vague or untrue. The reader considered life to be the cause and not the consequence of organization, and any attempt to grapple with its constitution as being futile.

*March 23rd.—Mr. WAGSTAFFE in the Chair.*

Mr. CAMPBELL read a paper on "The Phrenological System of Gall, with modifications introduced into it by Spurzheim and Castle, illustrated by the faculty of 'Courage.'"

## NOTES OF A CASE

OF

### REMOVAL OF THE SCAPULA.

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By WILLIAM MAC CORMAC.

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REMOVAL of the scapula is an operation at once so formidable and comparatively so rare, that the record even of a single case cannot fail to be of some interest.

The patient was a young woman, *æt.* 29, who until her present illness had enjoyed good health.

She was admitted under my care into the hospital on April 26th, 1876.

The only history obtained was that sixteen years before, she experienced pain in the shoulder, but not until seven years ago did she discover a tumour. A lump was then felt below the spine of the scapula, about a walnut in size. This tumour slowly and painlessly increased until the Christmas of 1875, when it had reached the bulk of a large orange, the patient being able, notwithstanding, to do her work without inconvenience.

Since then, however, the rate of increase has been very rapid and attended by great pain, so that the patient wears a most anxious appearance, as is well seen in the woodcut (Fig. 1).

On admission a rounded tumour, quite as big as a boy's head, was found to involve the whole scapula except the tips

of the acromion and coracoid processes. It bulged up into the neck beneath the trapezius, lay over the subclavian vessels, and filled the posterior half of the axilla, having clearly invaded the sub-scapular fossa. The surface of the new growth was covered by tensely stretched skin, which was adherent to the more prominent part of the tumour, and altered in colour. One or two enlarged lymphatic

FIG. 1.<sup>1</sup>

glands could be felt over the clavicle and in the axilla. From the upper to the lower border, the tumour measured twelve inches. It moved on the thorax freely enough, and seemed perfectly circumscribed. The sense of fluctuation was distinct at several points, so much so, as to suggest the

<sup>1</sup> The two drawings, Figs. 1 and 2, copied most carefully and accurately from photographs, represent the anterior and posterior aspects of the tumour.

presence of cysts, and everywhere the tumour felt very elastic, but more solid in some places than others. The movements of the shoulder-joint appeared to be but little interfered with.

Two exploratory punctures were made, but blood only escaped through the canula each time, and *per saltum*, showing the extreme vascularity of the growth.

FIG. 2.



The patient had been for some time getting weaker and more exhausted from the great pain she was suffering. At night she got very little rest, and was only able to lie down in the prone position. She most earnestly demanded an operation to afford her relief from pain, felt not only in the tumour, but stretching down the arm from pressure upon the brachial plexus.

The originally slow and painless increase of the tumour,

and its almost certain connection with the scapula, seemed to point to the growth being probably in the first instance cartilaginous, to which had been lately superadded some new form of growth more rapid in its development. I decided after consultation with my colleagues that the case was a proper one in which to interfere surgically.

In considering the steps of the operation, I thought it desirable to experiment with my assistants, beforehand, several times upon the dead subject, so that each of them might know exactly what to do, and when to do it.

It was clear that loss of blood was especially to be guarded against, as the patient was quite unfit to sustain any serious haemorrhage, in addition to the inevitable shock.

One most important detail towards effecting this is the sawing through of the clavicle, as a preliminary measure, just within the ligaments, which saves much time otherwise unavoidably lost in trying to disarticulate at the acromio-clavicular joint, whilst the operation is in progress, and the bleeding going on. Another important point is so to arrange the steps of the operation as to divide the subscapular artery quite towards the end, an assistant grasping the flap beforehand. This will prevent any bleeding taking place from this, by far the largest vessel divided.

The patient having been etherised, I proceeded to excise the scapula on May the 10th. The general plan of the incision was T-shaped, one line of the T extending along the upper part of the tumour from the acromion to its vertebral margin, and so planned as to overlie the clavicle near the point at which it was to be divided. The vertical incision was modified by the necessity of sacrificing a large portion of the skin, so that there were actually three superficial incisions nearly corresponding to, but within, the costal margins of the scapula; the triangular included portion of implicated skin being removed with the growth.

The superior incision was first made, the skin raised from the tumour, and the clavicle exposed. This was then divided just internal to the coracoid process with a small saw.

Next, the axillary flap was raised, the shoulder exposed, and the deltoid, with the subjacent capsule of the joint, divided near their attachments by a horizontal sweep of the knife.

It now remained to free the internal and lower borders of the tumour, and then catching hold of the inferior angle of the scapula, while an assistant supported the mass, to dissect the tumour rapidly out from below upwards. During this stage of the operation the subscapular artery can be felt, and readily grasped by another assistant before it is divided. No time need now be lost in separating the remaining connections of the scapula with the arm and clavicle, as this has previously been all but accomplished, and a few touches of the knife serve completely to separate the scapula and the small portion of clavicle attached to it.

In this case some little time was consumed in freeing the superior connections of the growth, which dipped deeply into the neck beneath the trapezius, and lay upon the sheath of the subclavian vessels, from which it required careful dissection. The subclavian artery being afterwards exposed in the wound for a length of nearly two inches. The enlarged glands were removed at the same time. The operation was, however, completed in fifteen minutes.

Scarcely any blood, not more than two ounces, was lost from first to last. The subclavian artery is easily accessible through the first incision, but it did not in this case need being compressed. Bulldog forceps were applied to the vessels as they were divided, and they were afterwards secured by catgut ligatures. The tumour after removal weighed within a fraction of seven pounds.

Although the operation was rapidly completed, and so little blood was lost, the patient seemed dying from shock on the operating table. Her limbs were bandaged, stimulant injections given, and she was made to breathe in vapour of ammonia. But it was a considerable time before it was deemed safe to remove her to bed. There was subsequently much sickness, and she was kept alive for the first forty-eight hours by nutrient stimulating enemata.

The after progress of the case need not be reported in detail. The dressings were made with strict regard to asepsis with carbolised oil lint covered by salicylic wool.

In a week the wound was suppurating healthily. In a fortnight the woman could sit up a little in bed, and her chief, indeed only, complaints throughout her convalescence

were of numbness in the hand, and of the smell of the carbolic acid used in the dressings.

She soon lost the anxious expression she formerly had, as is seen in the photograph taken after recovery (Fig. 8).

FIG. 8.<sup>1</sup>

The wound healed slowly, as there was a large granulating surface to cicatrize, due to the loss of skin. The patient left the hospital in August, the wound all but healed, and having a fair use of her arm.

The further history is an unfortunate one. The poor woman returned again in November, 1876, with evident return of the disease in the internal organs, and above the clavicle there was a growth as big as a large walnut. There was every reason to

<sup>1</sup> Fig. 8 is copied from a photograph taken shortly before the patient left the hospital.

believe there were extensive secondary deposits in the lungs. The lungs were dull on percussion, but the physical signs otherwise were obscure. The respiration was much oppressed, and there was most copious foetid expectoration.

She quietly and suddenly died on November 12th, having just before been sitting up in bed drinking a cup of tea.

On examination after removal the tumour was found to engage the entire scapula, with the exception of the glenoid cavity, the tip of the acromion, and the tip of the coracoid. The framework of the bone had to a large extent melted down or otherwise become continuous with the muco-gelatinous tumour structure which formed the bulk of the growth.

Both lungs were found to be infiltrated almost throughout with secondary growths, almost identical in appearance and structure with the original tumour. Some patches were found in the pectoral muscles, and a mass implicating the veins above the clavicle.

Dr. Charles has kindly prepared sections of the tumours, and of the recurrent growth, and recorded the appearance in the following report.

*Microscopic examination.*—“The tumour was of the nature of a myxoma. The cells varied much in form and size, some round, others angular, fusiform, or provided with anastomosing processes. There were also in a few places large nucleated cells, closely resembling those of a chondroma. The amount of the intercellular mucous substance also varied greatly in different parts of the tumour, in places being very abundant, and in others so scanty that the cells (chiefly the round ones) lay so closely packed together as to give the appearance of a round-celled sarcoma. Elastic fibres were to be seen here and there in the mucous substance.

“The *secondary growth in the lungs* was more or less of the same character as the tumour, and presented itself in the form of nodules, mostly of the size of a pea, which were thickly scattered through the lung substance. Each of these little nodules was made up of a series of encapsulated masses of mucous tissue, containing round cells of variable size (from  $\frac{1}{1800}$ th to  $\frac{1}{7000}$ th of an inch), and fusiform and branched cells. The capsules consisted mainly of condensed lung-tissue, but in many places it appeared as if the growth had

taken place in the larger vessels and smaller bronchial tubes, distending them so as to form investments for itself. (It may be mentioned here that prolongations of myxomatous tissue were traced in the veins leading away from the secondary growths that appeared near the seat of the original tumour.) The surrounding lung-tissue in many places showed signs of congestion, with abundant small-cell infiltration; and haemorrhagic patches, as well as a condition resembling red and grey hepatization, were frequent, particularly in the lower lobes.

"The microscopical examination of both the primary and secondary growths showed, therefore, chiefly the characters of myxoma, probably engrafted on a chondromatous base."

The points of interest in the case are, first, the clinical history, showing a growth apparently benign during a series of years, which caused no pain, very trifling, if any, inconvenience, and with a slow and gradual increase in bulk. This was I should think a cartilaginous outgrowth from the scapula. Then six months before admission, very rapid increase of size with pain set in. The previous chondroma had become a myxochondroma, and finally the myxoid growth quite predominated and obscured the traces of the cartilage base. A benign was transformed into a malignant growth.

The operation itself was full of interest. In the first place the risk of loss of blood was effectively guarded against and prevented. The experience on the dead subject showed the method employed to be a quick and facile one, and by exercising one's assistants beforehand on the dead body in the very manœuvres they were afterwards called on to perform upon the living, the operation passed quietly and quickly over, without hitch or delay of any kind.

The occurrence of such profound shock, without loss of blood, proves, I think, that the shock after great operations, disarticulation of the femur, for instance, is due to other causes besides mere loss of blood. The depression was in this case so profound as to cause for a time much apprehension for the immediate safety of the patient.

The unfortunate result was, I consider, in some degree attributable to the patient herself. She refused for some years, until the last moment in fact, all idea of permitting an

operation. Had one been earlier performed it might have proved more permanently successful. As it was the tumour had probably diffused itself, even before the operation was undertaken.

The incision over the upper margin of the clavicle, together with the preliminary division of that bone, facilitates the operation very much indeed, and admits besides of ready compression of the third stage of the subclavian if desirable. But the separation of the tumour from below upwards, and the late division of the subscapular artery, really ensure that the haemorrhage shall be under very complete control.

The extensive nature of the lung infiltration was such as to appear quite incompatible with life, yet the physical signs of such serious lung change, though often looked for, were not discovered, and the patient only complained of difficulty of breathing.



## INTRACRANIAL ANEURISMS.

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By THOMAS B. PEACOCK, M.D., F.R.C.P.,  
SENIOR PHYSICIAN TO THE HOSPITAL.

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### PART II.

*Continued from page 174.*

IN the former part of this paper, after briefly alluding to the earlier recorded instances in which this condition of the arteries of the brain had been met with, I gave the particulars of three original cases, two of which had occurred in my practice at the hospital, and one which had fallen under the notice of Dr. Bristowe. The former abstracts referred to the cases published prior to the appearance of Dr. Brinton's report on the subject in the 'Pathological Transactions' for 1852. To these I appended tabulated statements of cases published since the appearance of the important paper by Sir W. Gull in the 'Guy's Hospital Reports' for 1859. This collection of cases was intended to have been subjected to analysis with the hope of throwing some additional light on the pathology of the cerebral aneurisms, but I was prevented by illness from carrying out my intentions. I now propose to supply some of the deficiencies which were unavoidable in the former part of the paper.

The aneurisms in the collected cases occupied the following vessels :

The internal carotid artery in . . . . .	12 cases.
(Exclusive of two which were supposed to be of the same kind, in which the patients still survived when the reports were published, the disease having undergone a spontaneous cure in one of them, and in the other the disease being arrested by ligature of the common carotid artery.)	
The aneurism assumed the arterio-venous form in . . . . .	1 case.
It occupied the anterior cerebral artery in . . . . .	4 cases.
(Including one which was seated on the artery of the corpus callosum.)	
The anterior communicating artery . . . . .	5 "
The disease was seated in the middle cerebral or Sylvian artery or its branches in . . . . .	27 "
In the vertebral arteries in . . . . .	5 "
In the basilar artery in . . . . .	22 "
In the cerebellar arteries in . . . . .	3 "
In the posterior cerebral in . . . . .	6 "
(And on various arteries—the vertebral, inferior and anterior cerebellar, the right Sylvian, and the posterior communicating arteries of both sides.)	1 case.
Making a total of . . . . .	86 "

The relative frequency with which the different vessels are thus shown to be the seat of disease differs somewhat from what was found in some other collections of cases, though the difference is unimportant. Thus, Sir W. Gull, from an analysis of his own and Dr. Brinton's cases, amounting to 51 in number, supposed the basilar artery to be the vessel most frequently affected, that artery being aneurismal in 20 cases, and the middle cerebral, which in my own collection was the most commonly affected, being only the seat of disease in 15 cases. Dr. Bartholow found, on comparing his own cases and those of Lebert and Durand, that in 40 cases there were aneurisms of the basilar artery, and in 41 of the middle cerebral.

The only other noticeable fact is that, with the exception of the one case, in which various vessels were affected, there is, in my own tables, no case of aneurism of the posterior communicating artery, while Dr. Bartholow refers to 8 as reported by different writers. He has also shown that the arteries in the left side of the brain are more frequently affected than those of the right side. Thus, in 40 cases the disease was on the left side, and only 31 on the right. I find

\*this conclusion confirmed by my own collection in 27 cases in which the middle cerebral or its branches were affected; in 15 the aneurisms were situated on the artery of the left side of the brain, and in only 11 on that of the right side, and in 1 case both vessels were aneurismal. In 11 cases of aneurism of the internal carotid artery 4 occupied the vessel of the right side, and 7 that of the left.

The size which the aneurismal sac attained in the cases collected was as follows:

In the cases in which the internal carotid was the seat of disease the tumours were reported as small in	.	.	.	.	1 case.
The size of a horse bean in	.	.	.	.	1 ,
of a small nut in	.	.	.	.	1 ,
of a filbert in	.	.	.	.	1 ,
of a sparrow's egg in	.	.	.	.	1 ,
and of a bantam's egg in	.	.	.	.	1 ,
The aneurisms of the middle cerebral artery are described as small in	.	.	.	.	2 cases.
The size of peas in	.	.	.	.	2 ,
of large peas in	.	.	.	.	4 ,
of horsebeans in	.	.	.	.	8 ,
of a lentil in	.	.	.	.	1 case.
of hazel nuts in	.	.	.	.	2 cases.
of a small cherry	.	.	.	.	1 case.
of an acorn	.	.	.	.	1 ,
of a small hen's egg	.	.	.	.	1 ,
(And in two cases the sac was biloculate, or had projections from its surface.)					
In the cases which occupied the anterior communicating artery, the sacs are said to be about the size of small peas in	.	.	.	.	2 cases.
and of horse beans in	.	.	.	.	2 ,
The aneurisms of the basilar artery often consisted rather of general dilatation of the vessel, to the extent sometimes of being two or three times the natural size; when there was a distinct sac it is stated to have been—					
As large as a plum in	.	.	.	.	1 case.
as small beans in	.	.	.	.	2 cases.
as a haricot bean	.	.	.	.	1 case.
as a cherry	.	.	.	.	1 ,

In one case, in which a branch of the cerebellar artery was aneurismal, the sac which was embedded in the substance of the cerebellum was the size of two grains of wheat; and in another case of aneurism of one of the cerebellar arteries the tumour was as large as a nutmeg.

In aneurisms of the posterior cerebral artery the sac attained the size—

of a pigeon's egg in	.	.	.	.	1 case.
of a filbert in	.	.	.	.	1 "
of a large walnut in	.	.	.	.	1 "
of a hen's egg in	.	.	.	.	1 "

The symptoms caused by the intra-cranial aneurism are stated by Sir W. Gull not to have any specific character by which they can be diagnosed during life; and there is no doubt of the general correctness of this assertion, though in some cases diagnoses have been effected, and in one case successfully acted upon. When the tumours occupy the terminal portion of the internal carotid artery they frequently cause pressure on the nerves entering the orbit and on the optic nerves, and give rise to very marked symptoms, though there might be difficulty in distinguishing between the existence of an aneurism and any other kind of tumour having the same seat. In one instance, however, of this kind the nature of the disease was diagnosed eleven years before the death of the patient, and in another the affection was cured by the ligature of the common carotid artery. In this situation there sometimes occurs a communication between an aneurism of the carotid artery, where lying in the cavernous sinus, and the cavity of the sinus, producing well-marked symptoms, and one case of the kind is mentioned in my collection. When the basilar artery is aneurismal the tumour presses upon the pons and medulla and the nerves adjacent, and sometimes occasions atrophy, softening, or other secondary diseases of the parts. In some cases, however, tumours even of large size have given rise to very indefinite symptoms, and not unfrequently the first evidence of serious brain disease has been the occurrence of apoplectic symptoms, due to the rupture of the sac and the consequent extravasation of blood. These symptoms are not, however, always due to the rupture of the sac; in some cases an adjacent vessel has been found to have given way, and in others dilatation of small vessels in the substance of the brain have ruptured. When the sac gives way, though often there is a large outpouring of blood and the patient rapidly dies, in some cases, however, there is only a partial rupture, giving rise to slight coma and paralysis, and the patient recovers more or

less completely, sinking ultimately from a fresh rupture or from chronic brain disease, or from coincident diseases of some other organs—the heart, lungs, or kidneys. Death occurred from rupture of the sac in 17 of the 27 cases of aneurism of the middle cerebral artery, and in 14 of the 22 cases of aneurism of the basilar.

The relative frequency of aneurisms in the cerebral vessels in the two sexes and at different ages is shown in the accompanying table :

Under 15 years of age—Males	3.	Females	1.	Total	4.
15 to 20	"	8	"	2	" 5.
20 to 30	"	8	"	7	" 10.
30 to 40	"	8	"	7	" 15.
40 to 50	"	5	"	10	" 15.
50 to 60	"	10	"	10	" 20.
60 to 86	"	5	"	6	" 80.
	—	—	—	—	—
	86	48		79	

Sir W. Gull inferred that the cerebral aneurisms were nearly equally common in males as in females, stating that of 25 cases 13 occurred in males and 12 in females ; and Dr. Bartholow, from an analysis of the cases of Lebert and Durand, concluded that males were more frequently affected, the proportions being in males 61 cases and in females 42. Probably Sir W. Gull is right, and there is no essential distinction in this respect, though it will be seen that the cases collected by myself tend to show a greater liability to the disease in females. The most remarkable peculiarity as to age is that the cerebral aneurisms occur in early life much more frequently than the ordinary forms of aneurism. Thus, out of the 79 cases no less than 4 were in persons between thirteen and fifteen years of age, and 5 others between fifteen and twenty.

From the extreme tenuity of the coats of the cerebral vessels it is difficult to suppose that they should be capable of expansion so as to admit of the formation of a *true sacculated aneurism*, and it seems equally unlikely that the internal coats should be ruptured without the external also giving way, so that a *consecutive false aneurism* should be formed ; yet in several of the published cases it is stated that the three arterial tunics could be traced in the sacs, and in others it is reported

that the internal coats were wanting, though whether this was due to their having been originally ruptured or to the gradual expansion of the sac does not appear. In some cases the aneurism was apparently the result of accident, and in one the sac is stated to have been bounded by the pia mater, the whole of the arterial tunics being wanting in one part. In some cases the disease seems to have originated in embolism, the vessel being probably gradually expanded when the current of blood is restored in the obstructed vessel. If this be, as it very possibly may be, a frequent mode of origin, it will explain the large size which the sacs sometimes attain, even when springing from vessels of small calibre, for the fibrinous deposit would strengthen the walls of the vessel, and so enable them to be expanded to a greater extent than they would otherwise be without giving way. Not unfrequently the aneurisms seem to be caused by syphilis, and in this case the expansion is probably due to the vessels being involved in disease of the membranes of the brain. The other cerebral arteries are often extensively diseased in cases of aneurism, being thickened, atheromatous, and indurated, but in some cases the disease of the vessels is said to have been limited to the immediate seat of the aneurism. Often the large vessels—the aorta and its branches—are extensively diseased, and not unfrequently the heart also.

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#### ADDENDA.

In 1847 Mr. Corfe related two cases. In the first of these, a man, æt. 35, had suffered for four months from headache and giddiness, and was then suddenly taken with dimness of sight, and fell in the street, and was paralyzed, and died in nineteen days. An aneurism the size of a hazel-nut was found to arise from the internal carotid artery, and there was a clot in the lateral ventricle and softening of one corpus striatum.

In the second case a man, about 40 years of age, died suddenly, and an extravasation of blood was found at the base of the brain, which had proceeded from the rupture of an aneurism of the basilar artery, the size of a walnut.

The case exhibited by Dr. Eager at the Manchester Pathological Society in 1846 is apparently that referred to by Sir W. Gull, as quoted in a French journal under the name of Dr. Lager. It occurred on the basilar artery in a man, æt. 58, who had pains in the head, followed by paralysis of the right side of the face and hemiplegia, with which he died suddenly. The aneurism, about the size of a bean, had ruptured, giving rise to copious extravasation.



REPORT OF  
THE OBSTETRICAL DEPARTMENT.

By ROBERT CORY, M.B. CANTAB.

FROM the 1st of January, 1875, to the 31st of December, 1875, both dates inclusive, 1438 women were attended, Measrs. Whitehead, Edmunds, Joseph, and Rossiter having been successively the resident accoucheurs. Of these 1416 resulted in single births and 22 in twins; 7 of the former were cases of abortion.

In the following table the presentations are classified :

*Presentations.*

	Among the 1416 single births.	Among the 22 twin births.	Total.
Vertex	1879	33	1412
Breech	18	7	25
Superior extremities, includ- ing the shoulder	3	2	5
Inferior extremities	6	1	7
Face and forehead	3	0	3
Foot and hand	0	1	1
Abortions	7	0	7

## Of the 1438 cases attended,

214	were 1st labours.	37	were 10th labours.
243	" 2nd "	17	" 11th "
192	" 3rd "	9	" 12th "
198	" 4th "	7	" 13th "
145	" 5th "	3	" 14th "
147	" 6th "	1	was a 15th labour.
89	" 7th "	2	were 16th labours.
74	" 8th "	1	was a 17th labour.
58	" 9th "		

The forceps were used in 89 cases; the reasons given for their use are as follows:

Delay at the brim	20	$\left\{ \begin{array}{l} 2 \text{ from faulty position of heads.} \\ 1 \text{ " anteversion of uterus.} \\ 6 \text{ " large heads.} \\ 7 \text{ " contracted pelvis.} \\ 1 \text{ " tedious.} \\ 3 \text{ not stated.} \end{array} \right.$
	29	
	37	
	1	
	1	
	1	
	1	
	1	

There were 28 cases of primiparæ among these 89 cases, which gives a percentage of 31.5; the general percentage of primiparæ among the 1438 cases being 14.9. One maternal death occurred from haemorrhage, due, probably, to placenta prævia (see reported cases, No. 66).

Of the children, 51 were males and 37 females; the sex of one is not stated; 5 were stillborn, or 5.6 per cent. The cases in which these occurred were further complicated—in 1 by presentation of the lower extremities—in 1 by placenta prævia.

Version was resorted to in 9 cases for the following reasons:

Placenta prævia	.	.	.	.	.	.	.	3
Accidental haemorrhage	.	.	.	.	.	.	.	1
Contracted pelvis	.	.	.	.	.	.	.	1
Induction of labour after dilatation of cervix	.	.	.	.	.	.	.	1
Presentation of superior extremities	.	.	.	.	.	.	.	3

Six of the children were stillborn, and there were two maternal deaths; both of the latter were cases of placenta prævia (see reported cases, 314 and 536).

The breech presented eighteen times among the single births, giving a proportion of 1 in 78·3 cases. In 5 of these cases the children were stillborn, i. e. 1 child in every 3·6, or 27·7 per cent. One of these children had a large posterior encephalocele.

In the 3 cases of the presentation of the upper extremities occurring among the single births 2 of the children were stillborn.

The following table gives the particulars of the cases of placenta prævia :

No.	Ago.	Cofine- ment.	Sex.	Result to Child.	Treatment.	Result to Mother.
3304	25	2nd	F	D	Partial separation of placenta ; version	Recovery.
77	26	4th	M	L	Partial separation of placenta	Recovery.
128	26	7th	M	L	Partial separation of placenta ; dilatation of cervix ; version	Recovery.
314	31	4th	F	L	Partial separation of placenta ; dilatation of cervix ; version	Death.
536	33	10th	M	D	Partial separation of placenta ; version	Death.
822	21	2nd	M	L	No treatment	Recovery.
1081	31	3rd	M	L	...	Recovery.
1460	22	3rd	M.	D	Version	Recovery.

The following table gives the cases in which the uterus was injected with a solution of ferric chloride; they are altogether 12 in number, 11 of them made good recoveries. The one case in which death occurred was one of placenta prævia (see No. 536, reported cases). The solution used was the Liquor Ferri Perchloridi Fortior, and the strength mentioned in the table refers to this as mixed with water in the proportion named.

No.	Age.	Confine- ment.	Date.	Complication.	Result to Mother.	Strength of Solution.	Condition of Mother requiring its use.
3357	25	4	March 10	...	Good recovery	1 in 4	Clearing away clots. Ergot and cold ineffectual.
21	32	5	April 20	Forceps	Recovered without bad symptom	"	Clearing clots and introduction of hand into uterus ineffectual.
77	26	4	Feb. 1	Placenta previa	Good recovery	"	Ergot and introduction of hand into uterus failed to stop hemorrhage.
138	24	8	April 19	Twins	Slight tenderness over left ovary for a few days; recovery	"	Ergot and clearing away clot ineffectual.
167	21	1	April 17	Adherent placenta, forceps	Recovered without bad symptom	"	Severe hemorrhage.
202	25	2	March 6	Adherent placenta	Recovered without bad symptom	"	Ditto.
253	19	1	May 17	Forceps	No unfavorable symptom	"	Ditto.
452	29	7	May 8	Turning	Recovered	"	Ditto.
536	33	1	June 4	Placenta previa	Death	"	Ditto
786	26	8	Oct. 17	...	Recovered	Not stated	Ditto
885	41	11	Oct. 21	Forceps	Recovered	"	Ditto
1481	22	1	Nov. 28	Forceps	Recovered	"	Ditto

Five maternal deaths occurred during the year, or 3·5 per cent. ; this is less than half the death-rate of the two preceding years, and is accounted for by the absence of puerperal fever.

The following table gives an outline of the cases which are more fully reported elsewhere :

No.	Age.	Confine- ment.	Sex.	Result to Child.	Cause of Death.	Date of Confine- ment.	Date of Death.
66	35	9th	F	D	Accidental haemorrhage	May 2	May 2
314	34	4th	F	L	Placenta praevia	Mar. 11	Mar. 12
*370	41	11th	M	D	Accidental haemorrhage	May 6	May 6
461	35	2nd	F	L	Adherent placenta	Aug. 17	Aug. 17
*536	33	10th	M	D	Placenta praevia	July 24	July 24

\* Transfusion was tried in these cases.

Among the 1438 cases there were 22 cases of twins and 7 cases of abortion, hence the number of viable children born were  $1438 + 22 - 7 = 1453$ .

Among the viable children there were 53 stillborn, being in the proportion of 1 in 27·4, or 3·64 per cent.

The characters of the labours in which they occurred are given below :

Natural labours	.	.	.	.	.	.	.	25
Premature labours	.	.	.	.	.	.	.	7
Forceps cases	.	.	.	.	.	.	.	3
Version	.	.	.	.	.	.	.	4
Presentations of lower extremities	.	.	.	.	.	.	.	2
Breech presentations	.	.	.	.	.	.	.	5
Twins	.	.	.	.	.	.	.	3
Prolapse of cord	.	.	.	.	.	.	.	1
Accidental haemorrhage	.	.	.	.	.	.	.	2
Monster birth	.	.	.	.	.	.	.	1

The following table gives particulars of the cases of twin births.

No.	Age.	No. of Confinements.	Date of Birth.	Sex.	Result to Mother.		Result to Child.	Presentation.		Placenta.	
					1st.	2nd.		1st.	2nd.		
3342	34	6th	Jan. 27	F	F	R	L	L	Head	Breech	Separate.
3372	36	12th	Feb. 26	M	M	R	D	L	Head	Breech	Separate.
4404	26	6th	May 10	F	F	R	L	L	Head	Head	Single.
131	34	7th	March 28	M	M	R	L	L	Head	Head	Separate.
133	24	3rd	April 19	F	F	R	L	L	Head	Head	Single.
233	30	4th	March 13	M	F	R	L	L	Head	Head	Single.
254	33	7th	May 24	M	M	R	L	L	Natural	Natural	
285	37	4th	April 12	M	F	R	L	L	Head	Breech	Separate.
371	31	8th	April 30	M	M	R	L	L	Natural	Natural	Single.
372	38	8th	May 15	M	F	R	L	L	Head	Head	Separate.
392	22	1st	April 24	M	F	R	D	L	Arm	Breech	Separate.
414	30	9th	April 14	M	F	R	L	L	Head	Foot and hand	Single.
418	34	6th	June 24	F	F	R	L	L	Head	Hand	Single.
474	25	4th	June 10	M	F	R	L	L	Head	Feet	Single.
49	33	4th	June 9	M	F	R	L	L	Head	Head	
59	27	4th	July 21	F	F	R	L	L	Natural	Natural	
931	36	6th	Sept. 29	M	M	R	L	L	Head	Head	Single.
980	34	4th	Sept. 30	F	F	R	L	L	Head	Head	Single.
1132	26	5th	Nov. 25	F	F	R	L	L	Head	Head	Single.
1232	32	6th	Nov. 12	F	F	R	L	L	Natural	Natural	
1150	31	3rd	Dec. 16	F	M	R	L	L	Breech	Head	Single.
1345	25	4th	Dec. 10	F	F	R	D	L	Breech	Breech	Single.

It thus appears that only 3 cases of twins occurred in the months of July, August, September, and October, whereas there were 13 in the months of March, April, May, and June.

The placentæ were separate in 6 cases, single in 12; in 4 the condition is not recorded.

The children were—

In 5 cases, both males;  
 " 9 " " females;  
 " 8 " male and female.

There were 4 cases of spina bifida among the children.

The point of interest in one of these cases, 594, was the family history of malformations.

The mother's father's eldest son (the mother's half brother) had harelip and no roof to his mouth, and six fingers to each hand.

Child's father's brother's son (the child's paternal cousin) had talipes, and lastly, the child's own brother, aged two, his

immediate predecessor in the family, has harelip and a cleft which divides the gum, but does not extend into the palate.

The breech presented in three of these cases of malformation. The children all died.

#### *Cases of Maternal Deaths.*

66. *Accidental haemorrhage.*—S. G.—, st. 35, 9th confinement. When the obstetric clerk arrived he found the woman was suffering from severe haemorrhage, and immediately sent for the resident accoucheur. On his arrival he found the woman very pale, covered with a cold clammy sweat, pulse very feeble and fluttering (about 100), though at times too feeble to be perceptible. The woman was quite sensible, and complained of a dull aching pain in the abdomen and back, of faintness and constant shivering, of dimness of vision, and a sense of suffocation and of being unable to breathe. Her face had a sunken appearance and a very dark circle round the eyes. The breathing was hurried and sighing in character. The patient very restless.

She stated that she had been in labour a good many hours, but could not say how many, and that, although she knew she was losing a great deal of blood, she did not like to send for any help, because her pains "were not like proper ones," and she generally had lingering times. Examination showed that the haemorrhage had been most profuse, the bed being soaked. The head presented, and the os was fully dilated; forceps were at once applied, and very slight traction speedily completed labour. The birth of the child was followed by one large gush of blood, quickly followed by the placenta. The uterus, however, failed to contract, although means to that end were used; haemorrhage, however, ceased when the placenta was expelled.

From this time her pulse became more and more intermittent and feeble, and death appearing to be inevitable, Dr. Gervis was sent for in the hope that transfusion might be of service. The patient died before his arrival.

It afterwards transpired that she had been suffering from excessive haemorrhage for five weeks, but had persistently refused to seek advice.

314. *Placenta praevia.*—M. R.—, st. 34, 4th confinement. March 9th.—Patient states that she is now advanced about eight months in pregnancy, that two months ago, without known cause, she lost suddenly a great quantity of blood, and that since this she had been subject to periodical slight losses of blood at intervals of about a week. At 2 a.m. this morning the bleeding again returned severely, but she did not send to the hospital until 8.30 p.m. The resident accoucheur on his arrival found the patient pale, with a fairly good pulse; the bleeding had then ceased, and there were no labour pains. On examination he ascertained that the case was one of placenta praevia. The os uteri just admitted the tip of the fore-finger, and he at once separated as much of the placenta as he could reach; after this he advised the patient to remain in bed and to send up to the hospital in case haemorrhage returned or labour pains commenced.

The patient was seen twice daily until March 11th, when haemorrhage again occurred at 8.30 p.m., but no message was sent until 4.45 p.m. When seen at

5 p.m. she was blanched, with a feeble but regular pulse of 112, hurried respiration, and cold skin. Some brandy was then administered, and a hot-water bottle applied to the feet; after this the os was dilated by one of Barnes's bags. In three quarters of an hour the cervix was sufficiently dilated, and it was then found that the placenta completely covered the os. All of the placenta within the cervical zone was separated and an attempt made to apply the forceps unsuccessfully. Version was then resorted to, and a living female child easily extricated. The placenta came away readily and without haemorrhage; this occurred about 6.30 p.m. At 7 the woman was pulseless, and complained of feeling faint. More brandy was administered, after which the pulse returned, beating 160 per minute, feebly, but regularly. The uterus did not contract satisfactorily, and for two hours constantly tended to relax in spite of large and repeated doses of ergot and continued pressure, but at the end of this time, apparently by the use of ice, it became firmly and permanently contracted. It was not thought advisable to use the perchloride of iron, as there was no actual haemorrhage, and it might have increased the shock from which she suffered. At 11 the woman began to complain of much pain, which was limited to the epigastric region. Uterus firmly contracted. Pulse 120, and stronger than at 10 o'clock. No further haemorrhage occurred. The pain in the epigastrium continued, and the patient became anxious as to her state and very restless. At 1.10 a.m. she suddenly ceased to complain, and the pulse gradually became imperceptible, and she died quietly at 1.25 a.m.

379. *Accidental haemorrhage*.—J. R., st. 41, 11th confinement. The patient first noticed she was losing blood about 11 p.m. on May the 5th, but only having slight pains she did not send to the hospital until 3.30 a.m. on May 6th. When seen she appeared much exhausted, and complained of feeling faint. Her pulse was quick, small, and thready, and her forehead was bedewed with large drops of sweat. The os was found dilated to the size of a crown piece, and a slight loss of blood was going on. The membranes were ruptured and labour progressed satisfactorily. At 5.35 the child was born naturally, and during the pain which expelled it the uterus was held and pressed down upon. Immediately after the birth of the child a very large clot came away, and the placenta followed naturally in about ten minutes afterwards. The uterus did not contract at all satisfactorily, and as haemorrhage was still going on a solution of Liq. Ferri Perchlor. was injected, which at once arrested all haemorrhage, although the uterus did not contract. The patient did not appear to rally, the pulse became very weak, 140, skin cold and clammy, and the patient complained of faintness. Dr. Cory was then sent for by the resident accoucheur with a view to transfusion being performed as the only means of saving the patient's life. Mr. Wagstaffe returned with Dr. Cory, bringing his own apparatus with him. Some time was necessarily spent in obtaining the blood from the husband and desibrinating it. It was then mixed with milk, but the patient was breathing her last before the canula was introduced. A very small quantity of blood could be got to flow into the vein, and the patient died ten minutes afterwards without recovering consciousness.

536. *Placenta praevia*.—Jane D., st. 38, 1st confinement. From statements

made by people about the patient it seems that on the morning of July 24th she was taken suddenly with haemorrhage about two hours before application was made to the hospital for assistance. At a little before 11 a.m. the assistant obstetric clerk was sent for, and when he arrived a few minutes later he found the patient on the floor of the back kitchen, where she had fallen down. The patient was lying in a pool of blood and was senseless; her pulse could only just be felt at the wrist, and she was of a ghastly colour. The assistant obstetric clerk, finding that the vagina was at that moment plugged naturally by the coagula which had formed, did not make an examination, but sent to the hospital for the resident accoucheur; meanwhile he administered small doses of brandy-and-water to revive the patient. The resident accoucheur on his arrival found the woman in a state of collapse, and fearing immediate danger of death, he sent for Dr. Cory. Whilst waiting he removed all clots from the vagina, and found the source of the haemorrhage to be the cervical zone, from which the placenta was partially detached. The placenta was extensively attached to the anterior and left lateral portion of the cervix, and also slightly to the posterior; this he further separated as far as he could reach. The os was well dilated, so that quite three fingers could be inserted through the aperture. Upon this all haemorrhage ceased. The head presented. Dr. Cory having now arrived he turned by the bipolar method and quickly brought down a leg, and the child was born in less than half an hour, the patient remaining on the floor where she had at first fallen. The placenta soon followed, and the uterus immediately contracted. A solution of perchloride of iron was injected so as to make sure of no more bleeding. The child, a female, at full term, was dead.

From the time of delivery, at 1 p.m., up to the time of death the patient scarcely rallied. She took brandy-and-water, and half a pint of warm beef tea and half a pint of warm milk, and had no vomiting. At 2.48 p.m. she was much worse, and transfusion was proposed. By the time the instrument was ready and the vein in the arm opened the patient was no more. She died at 3.15 p.m. The patient had a drunken woman attending upon her. She was entirely neglected by her friends.

461. *Adherent placenta*.—Emma P.—, st. 35, 2nd confinement. During her pregnancy she had complained of a good deal of pain. She was rather desponding during the labour, but began to pick up after the birth of the child, which occurred naturally and in due time. The placenta did not come away properly, and obstetric clerk sent for resident accoucheur. On examining resident accoucheur found part of the placenta retained, but the uterus contracted upon it. He removed part of the placenta, but found a considerable portion firmly adherent. He sent for Dr. Cory; when he arrived the woman was very collapsed, and died five minutes after. There had been no large amount of haemorrhage.

#### *Case of Pemphigus following Delivery.*

210.—Ann F.—, st. 34, 6th confinement. Had five children previously; one living, three died in early infancy, one stillborn.

January 3rd.—The assistant obstetric clerk first saw case 12.30 a.m.; os not

much dilated; head presenting. From 3 a.m. until 7 p.m. no advance. The resident accoucheur was then sent for, and delivered with long forceps, the head being on the perineum, with occiput posterior. Child dead.

7th.—No enlargement of breasts; profuse diarrhoea; some tenderness of abdomen; temperature 103°. Ordered Ext. Opii Liq.  $\frac{1}{4}$  fl. oz., Acid. Sulph. Dil.  $\frac{1}{4}$  fl. oz., Decoct. Cinch.  $\frac{1}{2}$  j. 4tis horis. Vaginal discharge profuse and offensive; to be syringed with tepid Condy's fluid.

9th.—Diarrhoea continues; free perspiration; temperature 101.5°; pulse 108; tongue furred; abdomen somewhat tympanitic, tender on pressure in left iliac fossa. On the right thigh and leg scattered irregularly, but more on outer aspect, are a number of blebs as large as a pea and some larger; slight redness round base and containing pus. Ordered wine  $\frac{1}{2}$  jv, hot turpentine stupes to abdomen.

10th.—Diarrhoea ceased. Temperature 101.5°; pulse 110.

12th.—Abdomen no longer tender. Temperature 101.2°; pulse 116.

13th.—Diarrhoea returned; vaginal discharge still very offensive. Temperature 102°; pulse 116. Brandy  $\frac{1}{2}$  jv.

14th.—Diarrhoea better.

From this date she continued steadily to improve.

## MEDICAL REPORT.\*

1875.

TABLE I.—GENERAL STATEMENT.

		Males.	Females.	Total.
Number of patients in Hospital on Jan. 1st, 1875		61	70	131
"	Dec. 31st, 1875	...	...	...
"	admitted during the year 1875	...	...	1899
"	discharged	521	629	1150
"	died	155	94	249
Transferred to surgical wards		7	6	13

About 21 cases have not been tabulated, there being no corresponding bed-tickets. A repetition occurs in respect of two cases.

In many cases the result as to whether the patient was cured or relieved is not certain. A probable result has been given in those cases where no result was marked on the bed-tickets. In some cases a probable diagnosis has been given.

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\* Owing to Mr. Whitfield's sudden death some of the materials for this report have been mislaid, and it is consequently less complete than those of former years.—ED.

TABLE II.—*General Table of Diseases.*

DISEASE.	NUMBER OF CASES.			CURED.			RELIEVED.			UNCURED OR OTHER CAUSES.			DIED.			REMARKS, COMPLICATIONS, &c.		
	Total.	M.	F.	Total.	M.	F.	Total.	M.	F.	Total.	M.	F.	Total.	M.	F.	Total.	M.	F.
<b>GENERAL DISEASES.</b>																		
Ague	6	5	1	4	1	...	...	...	1	...	...	...	...	...	...	...	...	...
Miliary fever	1	1	...	1	1	...	1	1	...	1	...	...	...	...	...	...	...	...
Febrilena	17	6	11	6	10	...	1	1	...	1	...	...	...	...	...	...	...	...
Scarlet fever	58	28	30	23	28	1	...	...	...	...	...	...	4	2	2	One male, cured, tabulated under rheumatism also.	...	...
Measles	1	1	...	1	1	...	1	1	...	1	...	...	...	...	...	...	...	...
Enteric fever	39	23	16	19	14	...	1	1	...	1	...	...	...	...	4	2	...	...
Typhus fever	2	1	1	1	1	...	1	1	...	1	...	...	...	...	...	...	...	...
Influenza	1	1	...	1	1	...	1	1	...	1	...	...	...	...	...	...	...	...
Pyæmia, septicæmia	7	4	3	8	1	...	1	1	...	1	...	...	3	2	2	...	...	...
Erysipelas	45	21	24	19	24	...	1	1	...	1	...	...	1	...	...	...	...	...
Calcinitis	8	3	3	3	3	...	1	1	...	1	...	...	...	...	...	...	...	...
Rheumatism	157	76	82	61	64	8	...	...	12	2	3	4	3	1	3	One male, relieved, tabulated under pneumonia.	...	...
Syphilis	7	5	2	3	3	...	1	1	...	1	1	...	15	10	10	One male, cured, sent to ophthalmic surgeon.	...	...
Phthisis	81	45	36	1	...	...	1	26	22	8	4	...	8	1	1	One female, unrelieved, sent to surgeon.	...	...
Acute tuberculosis of fæces (tubercular)	4	3	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
Diabetes	7	4	3	...	...	1	...	1	3	1	...	...	2	1	1	...	...	...
Cancer	1	1	...	1	1	...	1	1	...	1	...	...	2	1	1	...	...	...
Gout	12	9	3	4	3	...	1	1	...	1	...	...	...	...	...	...	...	...
Anæmia, chlorosis	2	2	2	1	1	...	1	1	...	1	...	...	2	1	1	...	...	...
Diphtheria	7	4	3	4	3	...	1	1	...	1	...	...	2	1	1	...	...	...
Tubercular meningitis	2	2	2	2	2	...	1	1	...	1	...	...	2	1	1	...	...	...
Rheumatoid arthritis	8	4	4	2	2	...	1	1	...	1	...	...	2	1	1	...	...	...
Scarfætinal dropsey	14	10	4	6	3	...	1	1	...	1	...	...	8	2	1	...	...	...

This includes partial as well as general dropsy, viz oedema and anaæræa.

TABLE II—*continued.*

DISEASE.	NUMBER OF CASES.						REMARKS, COMPLICATIONS, &c.					
	Total.	M.	F.	M.	F.	M.	P.	M.	P.	M.	P.	DIED.
												UNRELIVED. OR OTHER CAUSES.
<b>CIRCULATORY SYSTEM.</b>												
Heart disease	65	35	30	...	...	1	17	16	2	1	16	12
Pericarditis	2	1	1	...	...	1	1	...	...	1	...	...
Aneurism	11	10	1	...	...	5	1	4	...	...	1	...
Disease of coronary arteries and aorta, and congestion of lungs	1	...	1	...	3	...	1	...	...	...	1	...
Thrombosis	4	...	4	...	3	...	1	...	...	...	...	...
<b>RESPIRATORY SYSTEM.</b>												
Asthma	1	...	1	...	7	11	7	18	2	1	6	6
Bronchitis and catarrh	53	22	31	...	...	9	1	1	...	...	14	5
Pneumonia	52	37	15	22	9	1	...	...	...	...	...	...
Cynanche tonsillaris	4	1	3	1	3	...	...	...	...	...	...	...
Obstruction of larynx	1	...	1	...	1	...	1	...	...	...	...	...
Sore throat	1	...	1	...	1	...	1	...	...	...	...	...
Croup	2	...	2	...	2	...	1	...	...	...	2	...
Hooping-cough	17	16	1	9	1	6	...	...	...	...	1	...
Pleurisy	...	...	...	...	...	...	...	...	...	...	...	...
Emphyema	6	8	3	1	1	1	1	1	1	1	2	...
Hemoptysis	14	11	3	8	3	...	3	...	1	1	2	...
Emphysema	4	4	...	...	...	...	2	...	...	...	1	...
Hydro-pneumo-thorax	2	2	...	...	...	...	...	...	...	...	...	...
<b>DIGESTIVE SYSTEM.</b>												
Hæmorrhoids	10	2	8	2	6	...	2	1	...	1	...	...
Biliary calculi	2	...	2	...	1	...	1	...	...	...	...	...

## URINARY SYSTEM.

**Renal disease and albuminuria**  
**Incontinence of urine**  
**Ulceration of bladder**

TABLE II—*continued.*

DISEASE.	NUMBER OF CASES.						REMARKS, COMPLICATIONS, &c.					
	Total	M.	F.	CURED.	RELIEVED.	UNRELIEVED. OR OTHER CAUSES.	DIED.	M.	F.	M.	F.	
<b>URINARY SYSTEM (continued).</b>												
Cystitis	1	1	...	1	...	1	...	...	...	...	...	...
Gravel	1	1	...	...	1	...	...	...	...	...	...	...
Edema of kidneys, dropped do., lungs	1	1	...	...	1	...	...	1	...	...	...	...
Uremic coma	1	1	...	...	1	...	...	...	...	...	...	...
<b>FEMALE ORGANS OF GENERA- TION.</b>												
Vulvar folliculitis	1	1	...	1	...	1	...	...	...	...	...	...
Vulico-vaginal fistula	2	2	...	2	...	2	...	...	2	...	...	...
Amenorrhoea	4	4	...	4	...	2	...	...	1	...	...	...
Dysmenorrhoea	14	14	...	8	...	10	...	1	...	...	...	...
Disease of os and cervix uteri	10	10	...	4	...	6	...	...	...	...	...	...
Metritis	1	1	...	1	...	1	...	...	...	...	...	...
Endometritis	5	5	...	2	...	3	...	...	3	...	...	...
Fibroid tumour of uterus	14	14	...	7	...	4	...	...	3	...	...	...
Subinvolution	6	6	...	4	...	2	...	1	...	...	...	...
Prolapsum uteri	2	2	...	2	...	1	...	1	...	...	...	...
Anteflexion	4	4	...	4	...	4	...	...	4	...	...	...
Retroflexion	14	14	...	1	...	1	...	1	...	14	...	...
Lateroflexion	1	1	...	5	...	2	...	...	3	...	...	...
Ruptured perineum	5	5	...	1	...	1	...	1	...	1	...	...
Anteversion	1	1	...	4	...	4	...	1	...	8	...	...
Retroversion	4	4	...	2	...	2	...	1	...	3	...	...
Pregnancy	1	1	...	1	...	1	...	1	...	1	...	1
Uterine conception	4	4	...	4	...	4	...	2	...	2	...	2
Polypus uteri	1	1	...	1	...	1	...	1	...	1	...	1

Menorrhagia	1	One case sent to surgeon.
Ovarian disease	1	
Abortion	1	
Carcinoma uteri	18	
Clinacteric	1	
Endocervicitis	1	
Atresia vaginis	1	
Paranovaritis	2	
Sloshing of vagina	1	
Vestico-vaginal fistula and	2	
ruptured perineum	1	
Hæmorrhage after abortion	1	
S.R.E.		
Eczema	8	
Erythema	1	
Ecthyma	8	
Tubercular elephantiasis	1	
E.R.B.		
Otorrhœa	1	
Poisons.		
Plumbium	10	
Hydrochloric acid	1	
Nitric acid	1	
Hydrazuryri oxyd. rubrum	1	
Oil of tar	1	
Chronic mercurial	1	
Opium	1	
Oxalic acid	2	
White precipitate	1	
Lin. camp. co.	1	
Mussels	1	
Crab	1	
Alcoholism	10	
Threatening delirium tremens	1	

TABLE II.—*continued.*

DISEASE.	NUMBER OF CASES.		CURED.		RELIEVED.		UNRELIEVED. OR OTHER CAUSES.		DIED.		REMARKS, COMPLICATIONS, &c.
	Total.	M.	F.	M.	F.	M.	F.	M.	F.	M.	
Submersion, drowning.	5	3	2	2	...	...	...	1	...	...	
Disease of lung and liver	1	1	...	2	...	...	...	2	...	...	
Absence of symptoms	2	...	2	...	...	...	...	1	...	...	
Diagnosis not recorded	107	63	45	18	18	13	13	4	9	27	11
Indefinite pains	2	1	1	2	1	1	1	1	1	1	1
Tumour of abdominal walls	1	1	1	1	1	1	1	1	1	1	1
Node	1	1	1	1	1	1	1	1	1	1	1
Abscess	6	2	4	1	1	1	1	1	1	1	1
Spinal curvature	4	1	3	1	1	1	1	2	1	1	1
Caries of spine	1	1	1	1	1	1	1	1	1	1	1
Disease of knee-joint	2	1	2	1	1	1	1	1	1	1	1
Weakness of legs	1	1	1	1	1	1	1	1	1	1	1
Marsasmus	2	1	1	1	1	1	1	1	1	1	1
Tumour of side	2	1	1	1	1	1	1	1	1	1	1
" axilla	1	1	1	1	1	1	1	1	1	1	1
Catarrh of fauces	1	1	1	1	1	1	1	1	1	1	1
Serofulous glands	1	1	1	1	1	1	1	1	1	1	1
Pelvic haematocele	1	1	1	1	1	1	1	1	1	1	1
Fits	2	1	2	1	1	1	1	1	1	1	1
Number of extremities	1	1	1	1	1	1	1	1	1	1	1
Malignant disease of pelvic organs	1	1	1	1	1	1	1	1	1	1	1
Atrophy	1	1	1	1	1	1	1	1	1	1	1

Pelvic tumour	.	.	2	...	2	...	2	...	...	...	...	...	One case sent to surgeon. One case sent to medical ward; this last was pelvic and abdominal tumours.
Partial aphasia, anasarca	.	.	1	1	1	...	1	...	...	...	1	...	This case was sent to surgeon.
Synovitis of ankle	.	.	1	...	1	...	1	...	...	...	1	...	
Chronic rheumatic effusion, left knee	.	.	1	...	1	...	1	...	...	...	1	...	
Debility	.	.	8	1	7	1	4	...	3	...	1	...	
" tubercle	.	.	1	1	...	...	...	...	...	...	1	...	
Premature labour or miscarriage	.	.	1	...	1	...	1	...	...	...	...	...	About seventh month.
Miscellaneous and doubtful cases	.	.	48	23	25	14	16	5	6	3	2	1	1

## CAUSES OF DEATH.

Total number 249.

						Number—	
						Male.	Female.
Anasarca	.	.	.	.	.	...	1
Croup	.	.	.	.	.	...	2
Cancer	.	.	.	.	.	...	1
"    of clavicle, mediastinum, liver, brain, &c.					.	1	...
"    [of many organs					.	...	1
Diphtheria	.	.	.	.	.	1	...
Erysipelas	.	.	.	.	.	1	...
Enteric fever	.	.	.	.	.	7	1
Fever, simple	.	.	.	.	.	...	1
"    pneumonia					.	...	1
Gout, albuminuria, uræmia	.	.	.	.	.	1	...
"    enlarged heart, enlarged kidneys					.	1	...
Pyæmia	.	.	.	.	.	8	2
Phthisis	.	.	.	.	.	19	12
Chronic phthisis, indurated liver, ascites	.	.	.	.	.	1	...
"    "    dilated heart					.	1	...
"    "    "    bronchitis					.	1	...
"    "    "    rheumatoid arthritis					.	...	1
Phthisis, constitutional syphilis, lupoid ulceration of genitals	.	.	.	.	.	...	1
Puerperal septicæmia	.	.	.	.	.	...	1
Rheumatism (acute), disease of heart, haematuria	.	.	.	.	.	...	1
Lardaceous disease of kidneys, syphilis	.	.	.	.	.	1	...
Syphilitic disease of brain	.	.	.	.	.	2	...
Scarlatina	.	.	.	.	.	1	1
Scarlatinal dropsey	.	.	.	.	.	4	1
Scarlatina, otitis, cerebral abscess	.	.	.	.	.	1	...
"    uræmia, gastro-enteritis, &c.					.	1	...
"    fracture right clavicle and first left rib, effusion					.	1	...
of blood into left pleura.					.	1	...
Acute tuberculosis	.	.	.	.	.	2	1
"    "    and meningitis					.	...	1
Tuberculosis, lungs, liver, spleen	.	.	.	.	.	1	...

		Number—	
		Male.	Female.
Ecthyma cachecticum ; no post-mortem	.	...	1
Tubercle of omentum, &c.	.	1	...
Acute ulceration of fauces, tuberculosis	.	1	...
Aneurism of middle cerebral arteries, apoplexy	.	1	...
Convulsions	.	1	...
Embolism of arteries of base of brain	.	1	...
Cerebral affection	.	...	1
Inflammation of brain	.	...	1
Heart disease, embolism, hemiplegia	.	1	...
"    " and softening of brain	.	...	1
Hydrocephalus	.	2	...
Tumour of brain	.	...	2
Haemorrhage into right corpus striatum, granular and contracted kidneys	.	1	...
Locomotor ataxy, cystitis	.	1	...
Acute disease of spinal cord, pyelitis, cystitis, and very large bed sore	.	1	...
Tubercular meningitis	.	2	...
Cancer of stomach and peritoneum	.	1	...
"    " pylorus	.	3	...
"    " stomach	.	1	...
"    " " and omentum	.	1	...
"    " pancreas	.	...	1
"    " hepatic glands	.	1	1
Intestinal obstruction from cancer of rectum	.	1	...
Cancer of uterus, peritonitis	.	...	2
Carcinoma uteri	.	...	1
Pelvic carcinoma	.	...	1
Malignant disease of pelvic viscera and peritoneum	.	...	1
"    " liver and pancreas	.	...	1
Cancer of right kidney and liver, ascites	.	1	...
Caries of dorsal vertebrae, peritonitis	.	1	...
Ulceration of intestines, pneumonia	.	1	...
Intussusception	.	...	1
Haematemesis, perforation of bowel	.	...	1
Cirrhosis of liver	.	3	2
"    " granular kidneys, &c.	.	1	...
"    " peritonitis	.	...	1
"    " hepatitis, haematemesis, chronic renal disease	.	1	...
Morbus hepatitis, cerebral disturbance	.	1	...
Abscesses of liver (hydatid ?) bursting into peritoneum	.	...	1
Jaundice	.	1	...
Ulceration of stomach	.	1	...
Enlarged spleen and lymphatic glands	.	1	...
Lymphadenoma	.	1	...
Ovarian tumour, peritonitis	.	...	1

		Number—
		Male.      Female.
Albuminuria	.	1      1
Bright's disease, aneurism of aorta	.	1      ...
Atrophy of left kidney, disease of right, gangrene of lung	.	1      ...
Malignant disease of kidney	.	1      ...
Chronic renal disease, adherent pericardium, effusion into right pleura	.	...      1
Disease of kidneys and disease of heart	.	...      1
Renal disease	.	3      1
Degeneration of kidney, obstruction of renal artery, ovarian tumour	.	...      1
Granular contracted kidneys	.	1      ...
Renal disease, bronchitis	.	1      ...
Disease of kidney, dropsey	.	...      1
Bright's disease, apoplexy	.	1      ...
Granular and contracted kidneys, hypertrophy of heart and aorta, pericarditis	.	1      ...
Granular kidneys	.	2      ...
Edema of kidneys and lungs	.	1      ...
Addison's disease	.	...      2
"      " tubercle	.	1      ...
Albuminuria, emphysema	.	1      ...
Bronchitis, morbus cordis	.	...      1
Bronchitis	.	...      1
"      anasarca	.	...      1
Chronic bronchitis, acute effusion into right pleura	.	1      ...
Subacute capillary bronchitis, phthisis	.	...      1
Chronic bronchitis, emphysema	.	2      ...
"      " dilated heart, acute pneumonia	.	...      1
Diseased spine and bronchitis	.	...      1
"      " chronic bronchitis, dilated heart	.	...      1
Pneumonia	.	6      1
Tubercular pneumonia	.	1      1
Pneumonia, albuminuria	.	1      ...
Pleuro-pneumonia	.	1      1
Broncho-pneumonia	.	3      2
Acute pneumonia, acute nephritis	.	1      ...
Pneumonia, emphysema, goitre	.	...      1
Pleurisy and broncho-pneumonia	.	...      1
Pleurisy	.	1      ...
Alcoholism, inflammation of lungs	.	1      ...
Inflammation of lungs, &c., after confinement	.	...      1
Adherent pericardium, bronchitis, albuminuria, rheumatism	.	1      ...
Empyema	.	2      1
Emphysema, &c.	.	1      ...
Hæmoptysis	.	1      ...
Disease of heart, aortic	.	1      ...

			Number—
			Male.      Female.
Disease of heart, aortic incompetence . . . . .			... . 1
"    "    "    "    valves, cancer of stomach . . . . .			1 . . . .
"    "    "    "    mitral . . . . .			1 . . 4
"    "    "    "    pulmonary valves . . . . .			1 . . . .
"    "    "    "    mitral, adherent pericardium . . . . .			... . 1
"    "    "    "    effusion in pleura . . . . .			1 . . . .
"    "    "    "    aortic and mitral . . . . .			1 . . . .
"    "    "    "    "    valves, ascites, renal affection . . . . .			... . 1
"    "    "    "    "    pulmonary apoplexy . . . . .			... . 1
"    "    "    "    "    false aneurism of brachial artery, pleuro-pneumonia . . . . .			1 . . . .
"    "    "    "    "    adherent pericardium dropsey . . . . .			1 . . . .
Morbus cordis et aortæ, adherent pericardium . . . . .			1 . . . .
Diseased heart, cirrhosis of liver, renal disease, bronchitis . . . . .			1 . . . .
Morbus cordis . . . . .			2 . . 6
"    "    granular kidney . . . . .			1 . . . .
"    "    cirrhosis . . . . .			1 . . . .
"    "    induration of liver, ascites, hydrothorax . . . . .			1 . . . .
"    "    cirrhosis of liver . . . . .			1 . . . .
Morbus cordis, renal, pulmonary apoplexy . . . . .			1 . . . .
Dilated heart, albuminuria, dropsey . . . . .			1 . . . .
Adherent pericardium, morbus cordis . . . . .			1 . . . .
Dropsey, hypertrophy, and dilatation of heart . . . . .			1 . . . .
Hæmorrhagic pericarditis, disease of aortic and mitral valves, rheumatism, acute . . . . .			1 . . . .
Pericarditis, bronchitis . . . . .			1 . . . .
Atheroma, gangrene . . . . .			1 . . . .
Aneurism of innominate and ascending aorta, erysipelas, gangrene of leg . . . . .			1 . . . .
Disease of coronary artery and aorta, congestion of lungs . . . . .			... . 1
Aneurism of arch of aorta, pneumonia . . . . .			1 . . . .
Thoracic aneurism . . . . .			1 . . . .
Aneurism . . . . .			1 . . . .
Atrophy . . . . .			1 . . . .
Cause uncertain, no post-mortem . . . . .			2 . . . .
Hydrophobia? . . . . .			1 . . . .
Opium poisoning . . . . .			1 . . . .
Caries of vertebrae, psoas abscess . . . . .			... . 1
Obesity (18 stone), bedsores . . . . .			... . 1
Strumous pyelitis, hypertrophy of heart, &c. . . . .			1 . . . .



# S U R G I C A L R E P O R T.

1875.

By SAMUEL OSBORN, F.R.C.S.,  
S U R G I C A L R E G I S T R A R.

## *General Statement.*

Number of beds . . . . .	220
patients in hospital January 1st, 1875 . .	192
" " admitted during the year 1875 . .	1579 { Males 928 Females 662
	1771 1590
" " in hospital December 31st, 1875 . .	181 { Males 96 Females 85
	181
" " treated during the year 1875 . .	1590
Discharged cured . . . . .	987
" relieved . . . . .	300
" on other grounds . . . . .	137
" dead . . . . .	168
	1590 . . . . .
	928 . . . . .
	662 . . . . .

Average number of deaths 10·4 per cent.  
" " days in hospital 35.

TABLE I.—*Abstract, showing Diseases, Injuries, &c., in Classes, according to authorised Nomenclature.*

	Total.			Ages.								Result.				Average stay in hospital in days.	
	M.	F.		5	10	15	20	30	40	50	60	+	C.	R.	U.	D.	
<b>I. GENERAL DISEASES.</b>																	
<i>Syphilis—</i>																	
Primary . . .	1	6				6	1					4		3			31
Secondary . . .	7	59				40	23	2	1			42	5	18	1		51
Congenital . . .		2	2									1			1		12
Chronic osteo-arthritis .	3	4					1	1	4		1	1	6				39
Rachitis . . .	3	1	3	1									1	3			28
Pyæmia . . .		1							1					1			25
<b>II. NERVOUS SYSTEM.</b>																	
<i>Diseases—</i>																	
Neuralgia . . .	6	2				1	1	1	1	1	2	1	3	3	2		26
Hysteria . . .	1	2					2	1				1	1	1			15
Tetanus . . .	1		1											1			1
Hydrophobia . . .	1		1											1			1
<i>Injuries—</i>																	
Concussion,																	
Cerebral . . .	25	5	4	4	4	3	4	7	1	1	2	25	1	3	1		10
Spinal . . .	2	2				1	1		2		3		3		1		19
Paralysis after injury .	1	1				1			1					2			8
<b>III. ORGANS OF SENSE.</b>																	
<i>Diseases—</i>																	
Gonorrhœal conjunctivitis .	1					1						1					88
Ulcer of cornea . . .	1											1					32
<i>Injuries—</i>																	
To eye . . .	10	3	2	1	1	4	2	2	1		5	6	2				17
Foreign bodies in external meatus . . .	1	1	2								2						2
<b>IV. ORGANS OF CIRCULATION.</b>																	
<i>Diseases—</i>																	
Aneurysm . . .	5	1	1			1		1	2	1		3	1		2		30
Epistaxis . . .		2								1	1	2					5
Hæmorrhages . . .	4					1		1	1		1	3		1			28
Varix . . .	7	10					2	3	7	3	2	8	5	2	2		30
Phlebitis . . .	1	1				1			1		2						29
Aneurysmal varix . . .	1						1					1					21
<b>V. RESPIRATORY ORGANS.</b>																	
Diphtheria . . .	1		1											1			2
Œdema of glottis . . .	1		1									1					24
Creup . . .	2	2												2			3
Impairment of speech .	1					1						1					28
Rupture of lung . . .		1					1					1					12
Laryngitis . . .	2	1				1	2						2	1			49
Pharyngitis . . .	1					1						1					20

TABLE I.—*Abstract, showing Diseases, Injuries, &c., in Classes, according to authorised Nomenclature*—continued.

	Total.	Ages.								Result.			Average stay in hospital in days.			
		M.	F.	5	10	15	20	30	40	50	60	+	C.	R.	U.	D.
<b>VI. DIGESTIVE ORGANS.</b>																
<i>Injuries—</i>																
<i>Hernia—</i>																
Reducible,																
Femoral	.	.	1	3	...	...	...	...	...	1	3	...	4	...	...	
Inguinal	.	.	11	2	3	1	...	4	...	2	3	...	13	...	...	
Ventral	.	.	1	1	...	...	...	...	1	1	...	1	1	...	12	
Umbilical	.	.	...	1	...	...	...	...	1	...	...	1	1	...	55	
Strangulated,																
Femoral	.	.	2	13	...	...	...	...	1	4	6	4	7	...	8	
Inguinal	.	.	5	...	...	...	1	...	2	...	2	...	...	5	3	
Umbilical	.	.	...	1	...	...	...	...	...	1	...	1	...	1	5	
Intestinal obstruction	.	.	2	1	...	...	...	...	1	...	2	...	...	...	22	
Rupture of intestine	.	1	...	...	...	...	...	1	...	...	1	...	1	0	0	
<i>Diseases—</i>																
Imperforate rectum	.	2	...	2	...	...	...	...	...	...	...	...	2	...	17	
Intussusception	.	...	1	1	...	...	...	...	...	...	...	...	1	...	3	
Stricture of oesophagus	.															
Simple	.	.	2	1	...	1	...	1	...	1	1	...	1	2	...	
Malignant	.	.	2	...	...	...	...	...	...	2	...	...	1	1	24	
Rectum—																
Fistula, fissure, abscess	14	9	...	...	3	7	5	5	2	1	18	2	2	1	40	
Stricture—																
Simple	.	.	1	9	...	1	...	3	5	1	...	1	2	4	3	
Malignant	.	.	1	3	...	...	1	1	2	...	...	2	2	...	52	
Anal prolapse	.	.	1	4	1	...	...	...	3	1	...	3	2	...	52	
<i>Hæmorrhoids—</i>																
Intero-external	.	2	2	...	...	...	2	1	...	1	2	1	...	1	25	
External	.	1	2	...	...	2	1	...	...	2	1	...	...	1	34	
<b>VII. GENITO—URINARY SYS.</b>																
<i>TM.</i>																
<i>Diseases—</i>																
Calculus	.	.	7	1	3	...	2	1	1	...	...	1	6	2	...	
Gonorrhœa	.		10	...	...	8	2	...	...	...	...	6	2	2	67	
Vaginal discharge	.		37	...	1	23	13	...	...	23	...	14	...	...	39	
Sores on vulva	.	.	17	...	...	10	6	1	...	10	...	7	...	...	61	
Menorrhagia	.	.	1	...	...	1	...	...	...	...	...	1	...	2	...	
Dysmenorrhœa	.	.	1	...	...	1	...	...	...	...	...	1	...	1	36	
Hypertrophy of nymphæ	.		2	...	...	2	...	...	2	...	...	2	...	...	103	
Irritability of bladder	1	1	...	...	...	...	1	1	...	1	1	...	1	1	19	
Ulceration of os uteri	.	1	...	...	...	1	...	1	...	...	1	...	1	...	43	
Prostatitis	.	.	1	...	...	1	...	1	...	...	1	...	1	...	19	
Retention	.	.	6	...	...	...	2	1	...	2	1	3	1	...	2	
Stricture	.	.	33	...	...	1	7	11	8	4	2	19	8	3	3	
Urinary fistula	.	.	5	...	...	2	3	...	...	1	3	...	1	1	70	
Urethral abscess	.	1	...	...	1	...	...	...	...	1	...	1	...	1	8	

TABLE I.—*Abstract, showing Diseases, Injuries, &c., in Classes, according to authorised Nomenclature*—continued.

	Total.		Ages.								Result.				Average stay in hospital in days.	
	M.	F.	5	10	15	20	30	40	50	60	+	C.	R.	U.	D.	
<b>GENITO-URINARY SYSTEM—</b>																
<i>continued.</i>																
Sores on penis . . . . .	5						5					5				25
Warts on penis . . . . .	1						1					1				35
Phimosis . . . . .	3					2			1			2	1			10
Adherent prepuce . . . . .	1	1											1			34
Epididymitis . . . . .	2					1	1					1	1			9
Orchitis . . . . .	6					4	2					6				15
Sarcocoele . . . . .	1						1					1				14
Hernia testis . . . . .	2		2									2				73
Tubercular testis . . . . .	1											1	1			20
Hydrocele . . . . .	7		1		2		2	1	1	4	3					19
Hæmatocoele . . . . .	2							2				1	1			44
Varicocele . . . . .	5				1	4						3	1	1		13
Cystitis . . . . .	1						1						1			26
Cancer of bladder . . . . .	1							1							1	32
Abscess in testicle . . . . .	3					1						2	3			47
Recto-urethral fistula . . . . .	1						1						1			101
<i>Injuries—</i>																
Lacerated kidney . . . . .	1							1				1				6
Ruptured urethra . . . . .	4			2			1	1				4				63
<b>VIII. GLANDULAR SYSTEM.</b>																
<i>Diseases—</i>																
Lymphangitis . . . . .	1						1					1				9
Hypertrophy . . . . .	1											1	1			54
Cancer . . . . .	1							1				1				35
Lymphadenitis . . . . .	2	5			1	1	4					1	5	1	1	33
Bronchocele . . . . .	4					8	1						4			56
Mammary abscess . . . . .	2					1	1					1	1			14
<b>IX. ORGANS OF LOCOMOTION.</b>																
<b>OSSEOUS SYSTEM.</b>																
<i>Diseases—</i>																
Periostitis . . . . .	3	3	1	1	2	2						2	3	1		43
Ostitis . . . . .	2		1		1			1				1	1			87
Necrosis . . . . .	36	6	2	4	7	6	14	5	1	8	26	9	8	4		74
Caries . . . . .	3	1	1	1	1							1	3	1		89
Abcess . . . . .	1					1							1			52
Neuralgia . . . . .	1					1							1			98
Ununited fracture . . . . .	5	1						2	3	1		2	2	2		82
Spontaneous fracture . . . . .	1						1					1				50
Exostosis . . . . .	1				1							1				88
<i>Joints—</i>																
Knee . . . . .	19	30	8	17	6	7	4	3	3	1	24	23	1	1		108
Hip . . . . .	32	11	13	16	6	8	4	1			10	24	4	5		79
Elbow . . . . .	10	3	1	2	1	1	3	2	1	1	6	5	1	1		66

TABLE I.—*Abstract, showing Diseases, Injuries, &c., in Classes, according to authorised Nomenclature—continued.*

	Total.		Ages.								Result.				Average stay in hospital in days.	
	M.	F.	5	10	15	20	30	40	50	60	+	C.	R.	U.	D.	
<b>ORGANS OF LOCOMOTION— continued.</b>																
Wrist	5	2		4			2	1			2	4	1			50
Ankle	4	4	1	2	2	2	1				2	4	1	1		98
Sacro-iliac	1						1					1				14
Jaw		1				1						1				34
Shoulder		1					1					1				66
Synovitis—																
Acute	22	8	1	2	4	6	8	5	2	2	26	3	1			29
Chronic	3	4	3	1	1	1	1	1	1			3	4			62
Disease of spine	3				2	1							3			45
Ankylosis of joints	5	12	4	2	4	4	1	2			7	9	1			50
Hysterical disease	3			1	1	1						2	1			18
Ganglion	1						1						1			14
Enlarged bursæ	10				3	1	3	1	2		10					27
Bursitis	3	7	1	2	3	2	2					9	1			20
Loose cartilage	1						1					1				3
<i>Injuries—</i>																
Sprains—																
Knee	1					1						1				9
Ankle	11	2				3	2	3	3	1	12	1				15
Spine	9		1	1	2		4	1			8	1				12
<i>Fractures—</i>																
Simple—																
Skull	8		1		1	1	4	1			2	1	5			5
Facial bones		1									1	1				7
Ribs	7					1	1	1	4		6	1				26
Spine	3							3								4
Pelvis	8	1	1			1	5	1	1	5						44
Clavicle	2		1								1	1	1			31
Sternum	1					1						1				48
Humerus	6	1	1	1	2		2	1			4	2	1			38
Radius and ulna	2	1			1	1	1				2	1				22
Radius	1	2		1				1			2	1				8
Ulna	2				1	1						2				29
Femur	37	18	16	9	4	2	1	1	4	5	13	48	3			45
Patella	10	7					5	5	6	1	16	1				39
Tibia and fibula	32	9	1	3	4	1	8	9	7	5	3	39	1	1		38
Tibia	19	6	5	2	4	2	3	3	2	3	1	24		1		28
Fibula	28	4	2	1		3	10	6	7	3	30	1	1			18
Bones of foot	3					1	1		1			3				29
Scapula	1	1				1	1				2					27
<i>Compound—</i>																
Skull	5	2	1	2		1	1	1		2	2			5		20
Facial bones	3	5	2	1	2	1	1				5	1		2		38
Femur	4		1		1		1	1			1	2		2		59
Tibia and fibula	12	1	1			3	4	3	1	1	8	1	4		73	

TABLE I.—*Abstract, showing Diseases, Injuries, &c., in Classes, according to authorised Nomenclature*—continued.

	Total.		Ages.									Result.				Average stay in hospital in days.
	M.	F.	5	10	15	20	30	40	50	60	+	C.	R.	U.	D.	
<b>ORGANS OF LOCOMOTION— continued.</b>																
Tibia . . . .	1		1								1					80
Humerus . . . .	3								1	1	1	2				46
Radius and ulna . . . .	1				1							1				1
Bones of hand . . . .	1						1									33
Bones of foot . . . .	3						1	1	1		3					70
<b>Dislocations—</b>																
Congenital, of hip . . . .		2			2							2				64
Pathological , , . .		2	1						1			2				22
<b>Traumatic—</b>																
Humerus . . . .	1	1							1	1		2				12
Spine . . . .		1					1					1				21
Scapula . . . .	1						1					1				56
Femur . . . .	1				1							1				28
Radius and ulna . . . .	3					2	1					1	2			23
Phalanges . . . .	2						2					2				50
Astragalus . . . .	1						1					1				41
Diastasis of sacro-iliac joint . . . .		1						1				1				30
Semilunar cartilage . . . .	1						1					1				55
Rupture of tendon . . . .	2						1				1	2				17
<b>X. CELLULAR TISSUE.</b>																
Inflammation . . . .	27	23	1	1	8	4	15	13	7	3	3	40	4	1	5	44
<b>Abscess—</b>																
Head and neck . . . .	7	5	1	1	3	2	2	2	1			7	5			28
Extremities . . . .	12	10	2	4	3	3	4	2	1	1	2	18	3		1	56
Trunk . . . .	12	7	3	1	4	1	3	2		2	8	15	8		1	45
Psoas and lumbar . . . .	5	3	1	1		1	4	1		1		2	5	1		73
Gangrene . . . .	4	1				1	1	1		2			5			32
<b>XI. CUTANEOUS SYSTEM.</b>																
<b>Diseases—</b>																
Eruptions . . . .	5	5				2	2	1	3	1	1	7	3			47
Ulcers . . . .	24	26	1	6	5	9	10	6	8	5	26	20	2	2		47
Sinus . . . .	2	2			1	1	1	1			3	1				41
Boil and carbuncle . . . .	6	2				1		2	3	2	8					29
In-growing toe-nail . . . .		1				1					1					25
<b>Injuries—</b>																
Burns . . . .	12	11	12			2	2	1	3	3	11		1	11		17
Scalds . . . .	9	16	19	4		1			1		18	2	1	9		10
<b>Wounds—</b>																
1. Incised—																
Head . . . .	1	1	2								1	1				14
Throat . . . .	6	2						1	2	5	7					22
Extremities . . . .	5	3	1	1	1	2	3				7	1				20

TABLE I.—*Abstract, showing Diseases, Injuries, &c., in Classes, according to authorised Nomenclature*—continued.

	Total.	Ages.										Result.				Average stay in hospital in days.
		M.	F.	5	10	15	20	30	40	50	60	+	C.	R.	U.	D.
<b>CUTANEOUS SYSTEM— continued.</b>																
2. Lacerated—																
Head	15	7	1	1		2	5	5	4	4	22					9
Extremities	17	3	1	3	4	8	5	2	2	19		1				47
Trunk	3	2	2	1		1	1				5					42
3. Contused—																
Head	4		2			1			1		4					3
Extremities	11	4	1	2	1	1	4	4	1	1	15					15
Trunk	21	8	6	3	2	2	8	5	1	2	26	1	2			11
4. Punctured—																
Thorax	1						1					1				95
Extremities	3	2				1	3	1			4		1			20
<b>XII. DEFORMITIES.</b>																
Talipes varus	3	5	6	1	1							8				98
" valgus	1	1				2						2				49
" equino-varus	1	5	2		4							6				38
" equinus	3		1	1		1						1	2			35
Contraction of plantar fascia	1				1							1				23
Torticollis	3	1		4								1	3			22
Contraction after burn	1		1									1				78
Deformities of nose	3				2	1						2	1			46
Knock-kneed	1	1	1			1						2				46
Deformities after injury	2					1	1					1	1			44
Harelip		1	1									1				64
Cleft palate	5	5	8	1	1							6	1	2	1	47
Spina bifida	1		1									1				8
Extroversio vesicæ	1		1										1			10
Malformation of fingers		1	1									1				4
<b>XIII. TUMOURES.</b>																
1. Innocent—																
Fibroma	1	3				1	1	1		1	1	1	1			37
Euchondroma	1					1						1				68
Lipoma	3	2				1	1	2	1		4	1				68
Adenoma		6				3	3				5	1				39
Angioma	1	4	4				1				2	2	1			16
Lymphangioma	1						1					1				164
Papilloma		5			1		1	3		4		1				29
Neuroma		1		1								1				69
Cysts—																
Sebaceous	2		1	1							2					20
Hæmatoma	1		1									1				28
Hydatid	2		1					1			2					19
Ovarian		5				1	1	3			1	1	3			21
Other	1	1	1			1					1	1				32

TABLE I.—*Abstract, showing Diseases, Injuries, &c., in Classes, according to authorised Nomenclature*—continued.

	Total.		Ages.									Result.				Average stay in hospital in days.
	M.	F.	5	10	15	20	30	40	50	60	+	C.	R.	U.	D.	
<b>TUMOURS—continued.</b>																
2. Semi-malignant—																
Sarcoma . . . . .	5	7	....	1	2	1	4	2	2	2	....	5	...	2	5	39
3. Malignant—																
Lymphomata . . . . .	1	1	1	....	1	....	....	....	....	....	....	....	....	1	1	28
Carcinoma . . . . .	4	16	1	....	....	1	2	10	5	1	7	1	5	7	41	
Epithelioma . . . . .	13	2	....	....	....	1	1	4	7	2	4	4	7	....	32	
Of doubtful nature . . . . .	5	9	....	1	1	3	3	3	3	3	....	2	9	3	....	45
<b>XIV. UNCLASSIFIED AND TRI- VIAL . . . . .</b>	20	8	5	....	2	3	5	5	5	3	....	12	2	5	9	7
<b>Total .....</b>	<b>928</b>	<b>662</b>	<b>173</b>	<b>123</b>	<b>121</b>	<b>218</b>	<b>311</b>	<b>239</b>	<b>191</b>	<b>134</b>	<b>80</b>	<b>887</b>	<b>800</b>	<b>137</b>	<b>166</b>	<b>35</b>

## SUMMARY OF DISEASES AND INJURIES, ETC., IN CLASSES.

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### I.—GENERAL DISEASES.

*Males.*—C. 7, R. 4, U. 8, D. 1.

*Females.*—C. 41, R. 8, U. 21, D. 2.

*Syphilis, primary.*—7: 1 M., chancre indurated, 3 weeks after connection, Pil Hyd. c. Opio, followed one week after admission by secondary symptoms treated by Pot. Iod. 6 F.; chancre indurated and amygdaloid glands in all cases; in 3 cases sore throat, in 2 secondary eruptions. Mercury given in all cases.

*Syphilis, secondary.*—66: 7 M., 3 with squamous eruptions and 4 with syphilitic ulceration. 59 F., 8 roseola eruptions, 23 with squamous eruptions, 29 with condylomata, and 11 with syphilitic ulceration. The fatal case was a very advanced form, patient insensible on admission, and but an imperfect history obtainable; hemiplegia of left side with perforation and necrosis of palate.

*Syphilis, congenital.*—One case was attributed, probably justly, to vaccination performed in Ireland; admitted with broken-down gummata; cured under the administration of mercury internally and externally. The second case, born of syphilitic parents, died from exhaustion consequent upon syphilitic ulceration.

*Chronic osteo-arthritis.*—7: disease in 4 cases being situated in knee, 3 in hip.

*General rachitis.*—4: two brothers were transferred the day after admission to the medical side with scarlet fever.

*Pyæmia.*—1: 1 M., no post mortem; multiple abscesses, disorganisation of wrist-joint as probable origin, high temperature, but no rigors.

### II.—NERVOUS SYSTEM.

*Males.*—C. 25, R. 2, U. 7, D. 3.

*Females.*—C. 7, R. 3, U. 2, D. 0.

#### DISEASES.

*Neuralgia.*—8: 6 M., 2 F., 1 with sciatica, 2 with painful stumps, 3 with neuralgia after former fracture, and 1 with neuralgia of fifth nerve, and 1 of internal cutaneous of both legs.

*Hysteria.*—3: 1 M., also of weak intellect, said to have had an injury to his head 8 months previously. 2 F.

*Tetanus.*—1: 1 M., st. 10, following contused and lacerated wound of hand 6 days before admission; stiffness of neck noticed 5 days after injury. Two middle fingers amputated and hydrate of chloral administered. Death 7 days after injury (see 'British Medical Journal,' Oct. 23rd, 1875).

*Hydrophobia.*—1: 1 M., st. 12 (see 'Lancet,' Oct. 23rd, 1875).

## INJURIES.

*Concussion.*—30 (cerebral): 25 M., in 8 cases there was probably fracture of skull, and the 1 fatal case was due to meningitis. 5 F.

*Concussion.*—4 (spinal): 2 M., in both cases numbness and inability to walk, but no paralysis of motion or sensation. 2 F., injury to lumbar region, pain and numbness in both legs; 1 case, occurring in a nurse with entire loss of power in both legs, was probably associated with a good deal of hysteria.

*Paralysis after injury.*—2: 1 M., 1 F., both of deltoid muscle.

## III.—ORGANS OF SENSE.

Males.—C. 6, R. 3, U. 2, D. 0.

Females.—C. 3, R. 3, U. 0, D. 0.

Diseases of the eye being treated in a special department, only injuries treated in the general wards are here enumerated, with two exceptions, which were patients admitted with other complaints, viz. gonorrhœa and disease of hip.

*Injuries to eye.*—13: 10 M., 3 F., in 3 cases only subconjunctival haemorrhage, 5 with haemorrhage into anterior chamber, and 3 with laceration of cornea and injury to other and deeper parts of the eye, but no case required extirpation of eyeball; 1 case of adhesions of eyelid to globe after scald.

*Foreign bodies in external meatus.*—2: 1 M., 1 F., in both cases foreign body removed under chloroform.

## IV.—ORGANS OF CIRCULATION.

Males.—C. 9, R. 4, U. 0, D. 5.

Females.—C. 9, R. 2, U. 2, D. 2.

## DISEASES.

*Aneurysm.*—6: 5 M., 4 true aneurysms and 1 false or traumatic aneurysm; 1 aneurysm of external iliac artery treated by flexion; patient, however, left hospital before cure was complete; 1 aneurysm of popliteal artery cured by digital compression after 12 hours; 1 aneurysm of ascending pharyngeal artery terminated fatally before carotid artery could be ligatured; 1 aneurysm of femoral artery cured after ligature of external iliac artery with catgut ligature, ligature left hanging out of wound (see 'Lancet,' May 27th, 1876). The traumatic aneurysm was of the lower part of brachial artery, for which the brachial artery was tied in its middle third, death occurred subsequently from heart disease. 1 F., traumatic aneurysm of ulna artery, digital compression of brachial artery having failed, tourniquet was applied also without effect; cure effected by ligature above and below aneurysmal dilatation.

*Epistaxis.*—2: 2 F., both following a blow, and anterior and posterior nares plugged in both cases.

*Hæmorrhages.*—4 M., 1 hæmorrhage from penis, 1 from bladder; 2 with hæmorrhagic diathesis, 1 after tooth-extraction and 1 with effusion into knee-joint.

*Varix.*—17: 7 M., 10 F., 6 admitted for hæmorrhage from ruptured varicose

vein and 11 with varicose veins, 6 of which had excision of vein performed after Mr. Marshall's method, and 4 were discharged relieved by pressure, and 1 operated upon by Mr. H. Lee's method of ligature and subcutaneous division, terminated fatally from phlebitis.

*Phlebitis.*—2: 1 M., 1 F.; both of internal saphena vein.

*Aneurysmal varix.*—1: 1 M., aneurysmal varix at the bifurcation of aorta with the left common iliac vein treated by instrumental compression of abdominal aorta, terminated fatally from gangrene of intestine due to obliteration of inferior mesenteric artery from the instrumental pressure (see 'Lancet,' March 6th, 1875, and 'Lancet,' Dec. 11th, 1875).

#### V.—RESPIRATORY ORGANS.

**Males.**—C. 2, R. 0, U. 1, D. 2.

**Females.**—C. 1, R. 1, U. 1, D. 2.

*Diphtheria and croup.*—3: 1 M., 2 F., in all cases tracheotomy was performed, but with fatal result.

*Œdema of glottis.*—1: 1 M., recovered after tracheotomy had been performed and artificial respiration kept up for twenty minutes.

*Impairment of speech.*—1: 1 F., relieved after removal of tonsils by guillotine and division of palato-glossi muscles.

*Rupture of lung.*—1: 1 F., haemoptysis after injury.

*Laryngitis.*—3: 2 M., 1 F., all with syphilitic history; the fatal case, after having tracheotomy successfully performed without chloroform, died 2 months after operation from an extension of disease downwards.

*Pharyngitis.*—1: 1 M., cured by Pot. Chlor.

#### VI.—DIGESTIVE SYSTEM.

**Males.**—C. 29, R. 2, U. 3, D. 18.

**Females.**—C. 22, R. 11, U. 8, D. 13.

#### INJURIES.

*Hernia, reducible.*—20: 13 M., 7 F., 13 of which were inguinal, 4 femoral, 2 ventral, and 1 umbilical; 10 had symptoms of strangulation, 6 were reduced by taxis, 4 by taxis under chloroform, and 7 reduced by ice-bag; 3 of the inguinal herniae in men were congenital, and the 2 cases occurring in females took place the one before, the other after menstrual activity; 2 ventral herniae, 1 in epigastric region reducible, the other over right iliac crest following pelvic abscesses. One umbilical hernia occurring in a woman with fat and lax abdominal wall.

*Strangulated (femoral).*—15: 2 M., 13 F., in 12 cases operated upon the sac was opened, 5 cases recovered and 7 died; in 3 the sac was not opened, 2 recovered, and 1 died, no post mortem being allowed, cause of death unknown; 11 were situated on the right side and 4 on the left; 2 died from broncho-pneumonia, 3 from peritonitis, and 2 from gangrene of intestine.

*Strangulated (inguinal).*—5: 5 M., in all cases the sac was opened, and all terminated fatally, 4 from peritonitis, and 1 from gangrene of intestine. In the

strangulated umbilical hernia the sac was also opened, and death resulted probably from peritonitis, but no post mortem allowed.

*Rupture of intestine.*—1: 1 M., due to the fall of companion upon him whilst wrestling, death from peritonitis and shock combined.

*Intestinal obstruction.*—2: 2 F., 1 due to obstruction from swallowing a piece of india rubber and the other to impacted faeces.

#### DISEASES.

*Imperforate rectum.*—2: 2 M., in both cases anus present and rectum found closed by a septum about half an inch up, which was brought into connection with external orifice.

*Intussusception.*—1: 1 F., aged 6 months; symptoms for 3 days; inflation tried, subsequently abdomen opened in median line; death one hour after.

*Stricture of oesophagus.*—5: 3 simple, 2 malignant; a boy, aged 11, with stricture due to former swallowing three years before of hydrochloric acid, came in with a piece of bacon impacted in the stricture; this having been removed under chloroform, stricture was dilated with bougies, and boy discharged.

The case of malignant stricture, there being virtually only 1, the man being admitted a second time for gastrotomy to be performed at his own request, was due to cancerous growth invading the pharynx. The operation was successfully performed by Mr. Sydney Jones, the patient dying subsequently from bronchitis. For detailed account see 'Lancet,' May 15th, 1875.

*Rectum and anus.*—Fistula 17: 13 M., 4 F. 4 blind external, 1 blind internal, 12 complete; in only 2 was there any history of consumption. Fissure 4: 4 F. 2 were forcibly split under chloroform, the other 2 treated by Conf. Sennæ internally and Arg. Nit. applied locally. Abscess 2: 1 M., 1 F., both resulted in fistulae. Stricture 14: 10 simple, 4 malignant; in all the simple cases there was a syphilitic history; 8 were treated with anti-syphilitic treatment and dilatation, the 2 fatal cases being due to pyæmia; 2 cases had colotomy performed, 1 died from peritonitis, the other and successful case was in a girl, aged 18, with hereditary syphilis, operated upon by Mr. Croft; 2 of the malignant cases had colotomy performed, both unsuccessfully. Prolapse 5: 1 M., 4 F., 3 of which underwent operation with the actual cautery. Haemorrhoids 7: 3 M., 4 F., 4 intero-external, 3 external, 4 underwent operation with clamp and cautery, 2 cured by ligature or removal with scissors, and 1 by internal remedies. The fatal case was due to embolism of arteries at the base of brain.

#### VII.—GENITO-URINARY SYSTEM.

##### DISEASES.

Males.—C. 65, R. 24, U. 5, D. 7.

Females.—C. 43, R. 5, U. 23, D. 0.

*Calculus.*—8: 7 M., lithotomy in 5 cases of vesical calculus, 1 urethral calculus removed by forceps, and 1 urethral calculus with extravasation of urine treated by perineal section. 1, aged 17, Camberwell, uric acid covered with phosphates, lithotomy, cured 64 days. 2, aged 12, Bermondsey, uric acid with phosphates,

lithotomy, cured 92 days. 3, aged 13, Lambeth, uric acid, extravasation of urine from impaction of calculus in urethra, perineal section, subsequent plastic operation for closure of fistula in perineum, cured 92 days. 4, aged 4, Lambeth, uric acid urethral calculus removed by forceps, cured 18 days. 5, aged 74, Montgomeryshire, uric acid with phosphates, lithotomy, cured 184 days (see 'Lancet,' July 8th, 1876). 6, aged 8, Maidenhead, uric acid, lithotomy, cured 49 days. 7, aged 2, Wandsworth, uric acid, lithotomy, cured 43 days. 1 F., aged 27, Camberwell, oxalate of lime, lithrotriptics relieved.

*Gonorrhœa and vaginal discharge.*—47 F., 7 cases only occurred in patients unfit for Magdalen ward; the 16 put under the head of unrelieved left the hospital without permission before cure was complete.

*Sores on vulva.*—17 F., all soft in character, and therefore not supposed to be syphilitic; 7 of these patients also left the hospital without permission.

*Menorrhagia.*—1: 1 F., transferred to Adelaide or special ward for diseases of women.

*Dysmenorrhœa.*—1: 1 F., R. 1.

*Hypertrophy of symphæ.*—2: 2 F., both occurred in women of low character, and cured by removal with galvanic cautery; 1 case was followed by erysipelas.

*Irritability of bladder.*—2: 1 M., 1 F., in both cases supposed stone, but none detected.

*Ulceration of os uteri.*—1: 1 F.

*Abcess in prostate.*—1: 1 M.

*Retention.*—6: 6 M., all of whom had stricture, 4 treated by cathetherism, 1 punctured above pubes by aspirator trochar died from peritonitis, and 1 treated by external urethrotomy died from cystitis and degeneration of kidneys.

*Stricture.*—33: 33 M., 19 of which were attributable to a former clap, and 8 to perineal injuries; 3 were associated with perineal abcess. 2 cases treated by external urethrotomy, 2 by internal urethrotomy, 1 by tapping above pubes, 1 by caustics, and the rest by cathetherism (see 'Med. Times and Gaz.,' Oct. 9th, 1875).

*Urinary fistula.*—5; 5 M., all due to stricture of urethra, and treated by cathetherism.

*Sores on penis.*—5: 5 M., all soft in character, 3 cured by W. W. D. 3 had inflammatory phymosis, 2 were of a sloughing character, and cured by opium and the topical application of strong sulphuric acid.

*Warts on penis.*—1: 1 M., non-syphilitic, removed by galvano-cautery.

*Phymosis.*—3: 3 M., all acquired, 1 slit up, 2 circumcised.

*Adherent prepuce.*—1: 1 M., also prolapsus recti, cured by circumcision.

*Epididymitis.*—2: 2 M., following gonorrhœa, and both of right side.

*Orchitis.*—6: 6 M., 4 due to gonorrhœa, 1 following tapping of hydrocele, and 1 due to injury.

*Sarcocele.*—1: 1 M., syphilitic history, and cured by Pot. Iod.

*Hernia testis.*—2: 2 M., both of the same patient, first of right one year ago,

then of left, attributed to blow, probably tubercular, cured by pressure and Ung. Hyd. nit. ox.

*Tubercular testis.*—1: 1 M., cured by Pot. Iod.

*Hydrocele.*—7: 7 M., 3 of right side, 2 of left, 1 encysted hydrocele of cord, and 1 double hydrocele. 2 tapped and injected, 3 simply tapped, 2 treated by pressure.

*Hematocoele.*—2: 2 M., in both history of injury, 1 cured by incision, and 1 relieved by ice.

*Varicocele.*—5: 5 M., all of left side. 2 relieved by palliative treatment, and 3 cured by pins, ligature, and subcutaneous division (see 'Brit. Med. Journ.', March 18th, 1876).

*Cystitis.*—1: 1 M., following gonorrhœa.

*Cancer of bladder.*—1: 1 M., of the colloid variety.

*Abscess in testicle.*—3: 3 M., after injury, 1 underwent castration, and in another testicle entirely disappeared by sloughing.

*Recto-urethral fistula.*—1: 1 M., relieved by operation.

#### INJURIES.

*Lacerated kidney.*—1: 1 M., haematuria following a fall of timber upon his loins.

*Ruptured urethra.*—4: 4 M., 2 cured by catheterism, and 2 by perineal section.

#### VIII.—GLANDULAR SYSTEM.

*Males.*—C. 4, R. 0, U. 0, D. 0.

*Females.*—C. 5, R. 6, U. 1, D. 0.

#### DISEASES.

*Hypertrophy.*—1: 1 F., of axillary glands after removal of breast, with neuralgia cured by removal of glands.

*Cancer.*—1: 1 M., of axillary glands after removal of both breasts for scirrhus.

*Lymphadenitis.*—7: 2 M., 5 F., 3 in groin, 1 in popliteal case, 2 in axilla, and 1 in middle of upper arm.

*Bronchocoele.*—4: 4 F., 2 from Wiltshire, 1 Bedford, and 1 Sussex. Increase in size at menstrual epoch. 2 tapped, and 1 of which was also injected with Tinct. Ferri Perchlor.

*Mammary abscess.*—2: 2 F., both intra-mammary during lactation.

#### IX.—ORGANS OF LOCOMOTION.

*Males.*—C. 280, R. 78, U. 9, D. 86.

*Females.*—C. 104, R. 51, U. 8, D. 12.

#### DISEASES.

*Periostitis.*—6: 3 M., 3 F., 4 due to injury, and disease in all cases situated in the leg; 1 acute case terminated fatally from pyæmia.

*Ostitis.*—2: 2 M., 1 of femur, and 1 of tibia, the latter probably syphilitic.

*Necrosis.*—42: 36 M., 6 F., 22 of tibia, 8 of stumps, 5 of femur, 3 of skull, 1 of jaw, 2 of palate, 2 of ilium, and 1 of each of the following bones: humerus, radius, ulna, phalanges, and fibula. Operations were performed in 26 cases and sequestra removed, amputation in 1 case. 5 were secondarily affected with erysipelas.

*Caries.*—4: 3 M., 1 F. Situation of disease: vertebrae 1, tibia 2, great toe 1. Amputation performed in 1 case, and gouging out of carious bone in 2.

*Abscess of bone.*—1: 1 M., lower end of tibia.

*Neuralgia of bone.*—1: 1 M., of tibia cured by trephining.

*Ununited fracture.*—6: 5 M., 1 F., 2 of tibia and fibula, 4 of humerus. 4 were cases of delayed union, and the other patient, twice admitted, had a seton passed between fragments, and then ends of bones resected, but without avail.

*Spontaneous fracture.*—1: 1 M., of femur, cause unknown, cured by splints.

*Exostosis.*—1: 1 F., of tibia, removed under chloroform.

#### JOINTS.

*Knee.*—49: 19 M., 30 F. In 5 cases amputation was performed, in 8 excision, 2 amputated after excision. In the 8 cases of excision origin of disease was synovial in 1 case, bone in 7. The fatal case was one in which amputation was performed subsequently to excision, death occurring from general tuberculosis.

*Hip.*—43: 32 M., 11 F., 4 cases underwent excision, 8 of which were in origin of disease bony, 1 synovial. In 1 case both hips were affected. The 5 fatal cases were due to the following causes: 2 from tuberculosis, 1 from diphtheria, 1 from pyæmia, and 1 from thrombosis of femoral vein after excision.

*Elbow.*—13: 10 M., 3 F., amputation performed in 3 cases, excision in 4, in 8 of which the origin of disease was probably synovial, 1 in bone. The fatal case was due to pyæmia following amputation for acute suppuration of joint.

*Wrist.*—7: 5 M., 2 F., excision in 2 cases, amputation in 1.

*Ankle.*—8: 4 M., 4 F. In 3 cases operations were performed, viz., amputation in upper third of tibia, Syme's amputation, and excision of joint surfaces; the latter terminated fatally from pyæmia.

*Sacro-iliac disease.*—1: 1 M.

*Jaw.*—1: 1 F., relieved by Pot. Iod.

*Shoulder.*—1: 1 F., treated by free incisions and draining tubes.

*Synovitis, acute.*—80: 22 M., 8 F., in 23 cases distinct history of injury, in 1 also rupture of external lateral ligament of knee, and 1 probably due to dislocation of semilunar cartilage. 1 was of wrist, 1 of ankle, the rest of knee.

*Synovitis, chronic.*—7: 3 M., 4 F., 4 of knee, 2 of ankle, and 1 of hip. 3 attributable to injury.

*Spine.*—3: 3 M., 2 with angular curvature, and 1 with double lateral curvature.

*Ankylosis of joints.*—17: 5 M., 12 F., 12 of knee, 2 of elbow, 1 of ankle, 1 of fingers, and 1 of jaw (see 'Med. Times and Gaz.', July 1st, 1876). Forcible flexion and extension performed under chloroform in 6 cases, 1 underwent

excision. The case of ankylosis of jaw was successfully operated upon by Mr. Mason after Rizzoli's method.

*Hysterical disease of joints.*—3: 3 F., 2 of knee, 1 of hip.

*Ganglion.*—1: 1 M., at the back of wrist, containing rice bodies, treated by puncture.

*Enlarged bursa.*—10: 10 F., 8 of bursa patella, 1 of bursa in connection with sartorius, and 1 on outer side of foot due to talipes. 4 were removed under chloroform. 6 occurred in servant girls from kneeling.

*Bursitis.*—10: 8 M., 7 F., 8 of bursa patella, 1 inflamed bunion, and 1 of bursa over olecranon.

*Loose cartilage.*—1: 1 M., relieved by Scott's dressing and strapping.

#### INJURIES.

*Sprains—knee.*—1: 1 M.

*Ankle.*—13: 11 M., 2 F.

*Spine.*—9: 9 M.

*Fractures—skull.*—Simple 8, compound 7. 18 M., 2 F., 8 being of vault, 4 of base and vault, and 3 of base. 2 with depression not followed by symptoms, 2 with merely fissure, and 1 with punctured wound from a fall upon some iron garden spikes died from cerebral and spinal meningitis. 9 others also died from cerebral meningitis.

*Facial bones.*—Simple 1, compound 8. 3 M., 6 F., 2 of nasal bones, 7 of inferior maxillary bone, 3 of the latter having also other fractures, 1 of 3 ribs and clavicle, 1 with fracture of radius, carpal, and superior maxillary bone, and the third with fracture of superior maxillary bone and both malar and nasal bones.

*Ribs.*—Simple 7. 7 M., no spitting of blood or any serious complication in any case. 2 only had slight emphysema.

*Spine.*—Simple 3. 3 M., all in dorsal region, death taking place in 2, 4, and 5 days respectively. 1 had also fracture of pelvis and 3 ribs. Only 1 had paralysis of motion and sensation in lower extremities.

*Pelvis.*—Simple 9. 8 M., 1 F., 1 with ruptured bladder and urethra, and 2 others required catheterism. 3 of ileum, 1 of ischium, and 2 of rami of ischium and pubes, the other doubtful.

*Clavicle.*—Simple 2. 2 M., both of left side.

*Sternum.*—Simple 1. 1 M., also injury to spine.

*Humerus.*—Simple 7, compound 3. 9 M., 1 F., 1 of the simple cases died from delirium tremens, and 2 of the compound required amputation, 1 of whom died from shock.

*Radius and ulna.*—Simple 3, compound 1. 3 M., 1 F.

*Radius.*—Simple 3: 1 M., 2 F., all in lower third.

*Ulna.*—Simple 2: 2 M., both of upper third.

*Femur.*—Simple 55, compound 4: 41 M., 18 F., 7 of which were intra-

capsular. 1 male, aged 31, double amputation, death from shock. 2, aged 5, amputation in upper third, recovery. 3, aged 18, death from haemorrhage and shock combined before amputation could be performed.

*Patella.*—Simple 17: 10 M., 7 F., 14 transversely by muscular action, and 3 stellate by direct violence.

*Tibia and fibula.*—Simple 41, compound 13: 44 M., 10 F. The cause of death of 1 simple fracture was delirium tremens. In the compound 3 had amputation performed below the knee, 2 amputation above the knee, 1 of the former who had also dislocation of tibia outwards proved fatal, the 2 latter proved fatal, 1 of whom had also amputation above elbow performed. 1 of the compound cases had necrosis of fractured extremities, which after being removed was discharged cured.

*Tibia.*—Simple 25, compound 1: 20 M., 6 F. 1 simple case died from delirium tremens.

*Fibula.*—Simple 32: 28 M., 4 F.

*Bones of foot.*—Simple 3, compound 3: 6 M., 1 of os calcis, 3 of phalanges, 1 of metatarsal bones, and 1 smash of foot, for which subastragaloïd amputation was performed.

*Scapula.*—Simple 2: 1 M., 1 F., both of inferior angle, 1 associated with fracture of clavicle.

*Bones of hand.*—Compound 1: 1 M., a railway porter, associated with severe laceration of hand; cold water irrigation first tried, subsequently 14 days after injury amputation in middle of forearm, followed after 6 days by tetanus; death from exhaustion. No post mortem.

*Dislocations—congenital.*—2: 2 F., both of the same patient. Birth natural; relieved by mechanical apparatus. Both hips affected.

*Pathological.*—2: 2 F., both of hip after joint disease.

*Traumatic—humerus.*—2: 1 M., 1 F., both subcoracoid. 1 reduced immediately after accident under chloroform; the other reduced without chloroform 16 days after injury.

*Spine.*—1: 1 F., in upper region of cervical vertebrae.

*Scapula.*—1: 1 M., downwards, associated with fracture of clavicle, radius, and ulna.

*Femur.*—1: 1 M., above sciatic notch, reduced under chloroform by manipulation.

*Radius and ulna.*—3: 3 M. 1, both bones backwards; 2, both bones backwards and inwards; 3, radius backwards, and fracture of internal condyle. Nos. 1 and 3 having occurred at 10 and 8 weeks previous were only partially relieved under chloroform. No 2 reduced the day after admission by extension under chloroform.

*Phalanges.*—2: 2 M., both compound, 1 of hand and 1 of foot.

*Astragalus.*—1: 1 M., outwards, associated with fracture of lower end of fibula.

*Diastasis of sacro-iliac joint.*—1: 1 M.

*Semilunar cartilage.*—1: 1 M., internal and associated with synovitis.

*Rupture of tendon.*—2: 2 M., 1 of biceps and 1 of ligamentum patellæ.

#### X.—CELLULAR TISSUE.

**Males.**—C. 47, R. 12, U. 2, D. 6.

**Females.**—C. 35, R. 8, U. 0, D. 6.

*Inflammation.*—50: 27 M., 23 F., all with the exception of 12 were of traumatic origin. 9 occurred in the head, 36 on the extremities, and 5 on the trunk; 4 of the latter were pelvic cellulitis occurring after confinement, 2 of whom died.

*Abscess—head and neck.*—12: 7 M., 5 F., 4 of which were alveolar from decayed teeth, and 1 followed scarlet fever.

*Extremities.*—22: 13 M., 10 F., all of which were of the lower extremity, the 1 fatal case being due to heart disease.

*Trunk.*—19: 12 M., 7 F., 8 of which were perineal; death in 1 case due to septicaemia.

*Psoas and lumbar.*—8: 5 M., 3 F., 3 were lumbar, 5 were psoas, 3 tapped by aspirator trochar.

*Gangrene.*—5: 4 M., 1 F., 1 of finger and 4 of toes; 1 inflammatory, 2 senile, 1 due to embolic plugging of femoral artery; 1 in a young man, aged 28, of symmetrical gangrene, cause unknown, probably inflammatory, became secondarily affected with tetanus, tracheotomy performed, died 2 days after seizure.

#### XI.—CUTANEOUS SYSTEM.

**Males.**—C. 114, R. 14, U. 2, D. 15.

**Females.**—C. 65, R. 14, U. 4, D. 12.

#### DISEASES.

*Eruptions.*—10: 5 M., 5 F., 8 were cases of eczema and 1 of scabies, 1 of syphilitic rupia, 1 of syphilitic lichen, 2 of elephantiasis, 1 of lupus, 1 of syphilitic maculae.

*Ulcers.*—50: 24 M., 26 F., 1 on lip probably syphilitic, 2 of anus, 3 of stump, 1 in a case of former double amputation; this case and 1 other were subjected to reamputation, 2 of cicatrices, and the remaining 42 were of the lower extremity, 6 being associated with varicose veins; 7 had distinct syphilitic history. 1 fatal case due to phthisis and 1 to exhaustion in a man, aged 61, after amputation below knee.

*Sinus.*—4: 2 M., 2 F., 1 of thigh, 1 in buttock, 1 in foot, and 1 after excision of knee.

*Boil and carbuncle.*—8: 6 M., 2 F., 3 situated on back, 2 on thigh, 2 on neck, and 1 on abdomen.

*In-growing toe-nail.*—1: 1 F., treated with solid nitrate of lead.

#### INJURIES.

*Burns.*—23: 12 M., 11 F., all the 11 fatal cases were severe burns of the trunk, occurring at the two extremes of life; 3 from shock, 5 from inflammation, and 3 from exhaustion. The treatment adopted in nearly all cases was the

immediate application of a paste formed by the mixture of whiting and olive oil in those of the second degree, whilst those above with poultices.

*Scalds.*—25: 9 M., 16 F.; the 9 fatal cases were extensive scalds, all with one exception of trunk, that being of face, arms, and hands, all occurring in young children from scalding water; 1 adult from hot beer. 4 died from shock, 3 from inflammation, and 2 from exhaustion. The treatment adopted was the same as for burns, viz. carron oil.

#### WOUNDS.

*Incised—head.*—2: 1 M., 1 F., slight concussion in one case.

*Throat.*—8: 6 M., 2 F., in 4 cases the larynx was opened, 2 through thyro-hyoid membrane, 1 through thyroid cartilage, and 1 through trachea. The 1 fatal case was due to shock.

*Extremities.*—8: 5 M., 3 F., 5 being of upper extremity and 3 of lower extremity; 1 case followed by erysipelas. Complications—1, division of superficial palmar arch and flexor tendons of 2 fingers; 2, of ulna artery and partially of ulnar nerve; 3, of superficialis volva.

*Lacerated—head.*—22: 15 M., 7 F. Periosteum stripped up in 4 cases, slight concussion in 2, erysipelas in 1. Temporal artery divided in 1 case.

*Extremities.*—20: 17 M., 8 F., 5 of the upper extremity, 15 of the lower extremity; 1 followed by erysipelas. Complications—1, division of biceps tendon; 2, amputation of 2 fingers; 3, amputation of last phalanx; 4, excision of ankle, on account of suppuration extending to joint.

*Trunk.*—5: 3 M., 2 F. Complications—1, peritonitis; 2, protrusion of intestine, castration; 3, protrusion of omentum, removal by clamp and actual cautery, subsequent closure followed by abdominal hernia.

*Contused—head.*—4: 4 M., all trivial cases.

*Extremities.*—15: 11 M., 4 F., all of lower extremity with one exception. Complications—1, absence of pulsation in arteries at wrist; 2, effusion into ankle-joints.

*Trunk.*—29: 21 M., 8 F. Complication—1, peritonitis.

*Punctured—thorax.*—1: 1 M., stab with a table knife in seventh intercostal space of right side. Hemothorax.

*Extremities.*—5: 3 M., 2 F., complications. 1, puncture of internal saphena vein with dissecting knife; 2, division of posterior interosseous nerve; 3, cellulitis, and subsequent death from pyæmia. 3 of upper extremity, 2 of lower extremity.

#### XII.—DEFORMITIES.

*Males.*—C. 6, R. 11, U. 1, D. 1.

*Females.*—C. 6, R. 18, U. 2, D. 1.

*Talipes varus.*—8: 3 M., 5 F., 6 were congenital, 2 paralytic. Tenotomy performed in 4 cases. Tendo-achillis in 2 cases, tibialis posticus in 2.

*Talipes valgus.*—2: 1 M., 1 F., both treated with mechanical apparatus.

*Talipes equino-varus.*—6: 1 M., 5 F. Tendo-achilles and tibialis posticus divided in 1 case, plantar fascia alone in another.

*Talipes equinus.*—3: 3 F., 1 congenital, 2 acquired. Tendo-achilles divided in 2 cases.

*Contraction of plantar fascia.*—1: 1 M., cured by division.

*Torticollis.*—4: 3 M., 1 F., all congenital and subjected to tenotomy.

*Contraction after burn.*—1: 1 M., relieved by division under chloroform.

*Deformities of nose.*—3: 3 F., following lupus. 2 were operated upon for the reconstruction of septum nasi.

*Knock-kneed.*—2: 1 M., 1 F., relieved by splints.

*Deformities after injury.*—2: 2 M., 1 following gunshot and 1 cellulitis; in the latter case amputation was performed.

*Harelip.*—1: 1 F., left side cured by operation.

*Cleft palate.*—10: 5 M., 5 F., 8 in soft and hard palate, 2 in soft alone. 8 were subjected to operation; 7 had harelip as well as cleft palate. 1 fatal case due to pneumonia.

*Spina bifida.*—1: 1 F., situated at the junction of dorsal and lumbar spine. Not operated upon.

*Extroversio-vesicae.*—1: 1 M., with epispadias.

*Deformity of hand.*—1: 1 M., extra thumb removed by amputation.

### XIII.—TUMOURS.

*Males.*—C. 12, R. 8, U. 16, D. 5.

*Females.*—C. 28, R. 11, U. 9, D. 14.

*Innocent—fibroma.*—4: 1 M., 3 F., 2 of buttock, 1 of jaw, 1 of vagina. 2 removed under chloroform.

*Enchondroma.*—1: 1 M., of tibia; removal.

*Lipoma.*—5: 3 M., 2 F., 4 of trunk, 1 case multiple, and 1 of leg. 4 were successfully operated upon, the multiple case relieved by Liq. Potassæ.

*Adenoma.*—6: 6 F., 5 of breast, 1 of neck. 2 had the whole breast removed, 1 of which was cystic in character, 2 had only tumour excised.

*Angioma.*—5: 1 M., 4 F., 3 on labia, 1 on leg, and 1 on head. 2 were operated upon.

*Lymphangioma.*—1: 1 M., of leg and scrotum, shown before Pathological Society (see 'Lancet,' Jan. 23rd, 1875).

*Papilloma.*—5: 5 F., 3 of mouth, 1 of rectum, 1 of labia. All cases operated upon. The 1 of rectum terminated fatally from peritonitis.

*Neuroma.*—1: 1 F., in leg, probably in connection with musculo-cutaneous.

*Cysts—sebaceous.*—2: 2 M., cured by removal.

*Hæmatoma.*—1: 1 M., following injury to thoracic wall.

*Hydatid.*—2: 2 M., both of trunk; removal. 1 M., aged 45 (see 'Lancet' June 5th, 1875).

*Ovarian.*—5: 5 F., all married women. Operation in 3 cases, all of which terminated fatally.

*Other cysts.*—2: 1 M., 1 F., 1 being hydrocele of neck, the other situated in chest wall.

*Semi-malignant—sarcoma.*—12: 5 M., 7 F., 4 were epulis of jaw, 3 underwent removal; 1 in which the whole superior maxillary bone was removed died from exhaustion. 1 of testicle died after castration. 1 of melanotic variety growing on old mole; removal. 1 of buttock, successfully removed. 3 of leg and 1 of arm were all treated by amputation, the 3 former terminating fatally.

*Malignant—lymphomata.*—2: 1 M., 1 F., both of cervical glands. 1 died from asphyxia before tracheotomy was performed.

*Carcinoma.*—20: 4 M., 16 F., 13 scirrhus of breast, 1 occurring in a man; 9 underwent operation. 1 in glands after amputation of breast, and 1 case of scirrhus of back; 2 malignant polypi; 1 cancer of coccygeal gland (see specimen in Museum of the Royal College of Surgeons, 1473 A); 2 of the soft or encephaloid variety, both of which died, 1 from pyæmia after operation, the other from heart disease.

*Epithelioma.*—15: 13 M., 2 F., 7 of tongue, 1 of bladder, 1 of vagina, 3 of lip, 1 of penis, 1 of cheek, 1 of toe. 5 underwent operation, 1 amputation of penis.

*Of doubtful nature.*—14: 5 M., 9 F., 5 of breast, 1 of spermatic cord, 4 in neck, 1 in abdomen, 3 in leg.

#### XIV.—UNCLASSIFIED AND TRIVIAL.

*Males.*—C. 8, R. 1, U. 4, D. 7.

*Females.*—C. 4, R. 1, U. 1, D. 2.

Six with medical ailments, 9 brought in dead, 8 with no ailment, and 5 not registered.

#### COMPLICATIONS.

##### ERYSIPELAS.

During the year 40 surgical cases were attacked with erysipelas, 25 of whom were males, 15 females. 18 occurred after operations, the remaining 27 in suppurating wounds. The following table shows the numbers affected in each month and in each ward.

Month.	Edward.	Leopold.	Alexan- dra.	Eliza- beth.	Albert.	Victoria.	Henry.	Total.
January .	...	...	1	2	...	...	...	3
February .	...	1	...	...	...	...	...	1
March .	1	1	1	1	...	...	...	4
April .	2	...	...	1	...	2	2	7
May .	2	...	...	...	2	1	...	5
June .	...	2	1	1	...	...	...	4
July .	2	2	1	1	...	...	...	6
August .	...	...	...	...	...	1	...	1
September .	1	...	...	...	...	1	...	2
October .	1	1	...	1	...	...	...	3
November .	3	...	...	...	...	...	...	3
December .	...	...	...	1	...	...	...	1
<b>Totals .</b>	<b>12</b>	<b>7</b>	<b>4</b>	<b>8</b>	<b>2</b>	<b>5</b>	<b>2</b>	<b>40</b>

TABLE II.—*Pyæmia,*

Of which there were 15 cases during the year, the particulars of which are given in the subjoined table.

No.	Ward, date of admission.	Age.	Primary disease or injury.	Operation.	Date of operation.	Onset of pyæmia.	Symptoms in evidence of pyæmia.	Date of death.	Remarks.
1	Edward 10, Jan. 18	44	Fibrous stricture of rectum	For fistula	Jan. 27	Feb. 1	Rigors, and fluctuating temperature	Feb. 6	No indications of pyæmia to be found post-mortem.
2	Elizabeth 16, April 1	33	Syphilitic stricture of rectum	...	...	June 9	Rigors, diarrhea, high temperature	June 14	Pelvic phlebitis; pyæmic abscesses in lungs and spleen; acute peritonitis
3	Leopold 14, Dec. 26	46	Perineal abscess and stricture of urethra	...	...	Jan. 10	No rigors; fluctuation of temperature and profuse perspiration	Jan. 16	Phlebitis of vein near neck of bladder; abscesses in lungs; pus in ankle; diffuse suppuration over back of hand.
4	Leopold 10, Jan. 25	23	Stricture of urethra	...	...	Jan. 31	Rigors, diarrhea	Feb. 2	Phlebitis of veins at neck of bladder; inflammation of knee-joint; acute pericarditis; abscesses in heart and lungs.
5	Leopold 7, Feb. 26	40	Urinary fistula	...	...	May 19	No rigors, fluctuating temperature	May 21	No post-mortem.
6	Leopold 20, Nov. 8	7	Acute periostitis	Amputation above knee-joint	Nov. 29	Dec. 6	Fluctuating temperature, abscesses, and suppuration in knee-joint	Dec. 10	No post-mortem.

7	Alexandra 10, Oct. 11	10	Disease of hip Incision	Oct. 16	Oct. 18	No rigors, high tempera- ture, cough, with loud mucous rales	Oct. 20	No post-mortem.
8	Edward 27, Sept. 11	30	Acute suppura- tion of above elbow	Sept. 20	Sept. 25	Rigors and profuse perspira- tion	Sept. 29	Enlarged spleen; pleural echymoses; no ab- scesses.
9	Alexander 9, Sept. 24	35	Disease of an- kle	Oct. 16	Oct. 19	Rigors, high temperature, diarrhaea	Oct. 22	No post-mortem.
10	Albert 3, May 6	35	Acute syno- vitis of knee	May 29	June 2	Rigors and fluctuating tem- perature; sloughing of stamp	June 20	Softened clot in femoral vein; embolic abscesses in lungs and spleen.
11	Elizabeth 2, Sept. 6	30	Cystic tumour of breast	Sept. 21	Oct. 18	Rigors, sickness, and high temperature	Oct. 21	No post-mortem.
12	Elizabeth 30, Dec. 8	66	Sarcoma tibia	Dec. 10	Dec. 15	Rigors, delirium, and fluctu- ating temperature	Dec. 24	Thrombosis of femoral vein; acute meningitis; pyemic abscesses.
13	Albert 22, Mar. 25	43	Punctured wound of arm and cellulitis Cellulitis of arm	April 8	April 11	Rigors, high temperature	May 13	Double purulent pleurisy; inflammation of left knee-joint.
14	William 8, May 29	46	Incisions	May 30	May 31	No rigors, high tempera- ture, with delirium	June 14	Erysipelas of thigh; en- larged spleen; dark fluid blood.
15	Victoria 8, Sept. 20	21	Compound fracture of inferior maxilla	...	Sept. 29	Convulsions and high tem- perature	Oct. 1	Abscesses in lung from phlebitis of internal jugular vein.

TABLE III.—*Surgical Operations, 1875.*

SURGICAL OPERATIONS.	Total.	Sex.		Ages.						Result.						
		M.	F.	5	10	15	20	30	40	50	60	+	C.	R.	U.	D.
<b>On ARTERIES.</b>																
Digital compression . . . . .	2	1	1				1			1				1	1	
ligature . . . . .	3	2	1				1			1	1			2	1	
<b>On VEINS.</b>																
Removal of haemorrhoids . . . . .	5	3	2						1	1	2		1	4		1
Obliteration of varicose veins . . . . .	8	2	6						2	1	3	2		7		1
Ditto, in varicocele . . . . .	3	3						1	2					3		
<b>On JOINTS.</b>																
<i>Reduction of dislocations—</i>																
a. Shoulder . . . . .	2	1	1							1	1			2		
b. Elbow . . . . .	3	3							2	1				1	2	
c. Hip . . . . .	1	1					1							1		
<i>Excision of joints—</i>																
a. Elbow . . . . .	6	5	1	1	1	1		2	1				3	3		
b. Hip . . . . .	12	9	3	7	2	2	1						3	6		3
c. Knee . . . . .	11	5	6	1	1	2	5	2					5	5	1	
d. Ankle . . . . .	4	3	1	1	1		1		1				2	1		1
e. Wrist . . . . .	3	3				2			1				1	2		
<b>On BONES.</b>																
<i>Removal of dead bone—</i>																
Radius . . . . .	2	2					1	1					1	1		
Metacarpus and phalanges . . . . .	3	2	1	1	1			1					2	1		
Femur . . . . .	2	2				1		1					1		1	
Tibia . . . . .	16	15	1	1	2	4	3	4				1	1	12	3	1
Fibula . . . . .	1	1					1						1			
Os calcis . . . . .	1	1							1					1		
Trephining of bone . . . . .	1	1						1						1		
For ununited fracture . . . . .	3	3							3						3	
Division of lower jaw . . . . .	1	1					1			1				1		
Subcutaneous division of femora . . . . .	1	1					1						1			
<b>AMPUTATIONS.</b>																
<i>1. For severe injury—</i>																
a. Arm . . . . .	3	3							2			1	2		1	
b. Hand . . . . .	1	1						1						1		
c. Thigh . . . . .	4	4		1	1				1	1			2		2	
d. Leg . . . . .	6	6			1			2	2			1	5		1	
e. Double . . . . .	8	3							3				2		1	
f. Fingers . . . . .	5	4	1	1	1		1	1	1				3		2	
g. Toe . . . . .	2	2					1		1	1			2			
<i>2. For disease—</i>																
a. Above elbow . . . . .	4	3	1				1	1		1	1		3		1	
b. Forearm . . . . .	1	1								1			1			

TABLE III—*continued.*

SURGICAL OPERATIONS.	Total.	Sex.		Ages.								Result.				
		M.	F.	5	10	15	20	30	40	50	60	+	C.	R.	U.	D.
<b>AMPUTATIONS (continued).</b>																
c. Fingers	1	1	1	1									1			
d. Hip-joint	1	1														1
e. Thigh	9	4	5	1	1	2	2	2					3	1		5
f. Through knee-joint	2	1	1	1	1								2			
g. Below knee	6	4	2			2		1		2	1	5				1
h. Syme's	1	1							1				1			
i. Double	1	1							1				1			
j. Subastragaloïd	1	1					1					1				
<b>REMOVAL OF CALCULI.</b>																
Lithotomy	6	6	4			1						1	5	1		
<b>INCISIONS.</b>																
For cicatrix from burn	2	2	1	1									2			
Loose cartilage	1	1						1					1			
Tracheotomy	7	5	2	5				1	1				1			6
Gastrotomy	1	1											1			1
Colotomy	3	1	2						1	2						3
Enterotomy	1	1	1										1			
For strangulated hernia	23	8	15				3	1	6	6	7	8	1		14	
For prolapsed rectum	3	3							2	1			3			
For imperforate rectum	2	2	2										2			
Removal of prepuce	3	3				2			1				2	1		
For impediment in speech	1	1				1							1			
Perineal section	6	6			2	1	1			2		2	2	2		2
Ovariotomy	3	3									3				3	
Rhinoplasty	2	2				2							2			
Cleft palate	1	1				1							1			
Harelip	7	3	4	7									5	1	1	
Castration	2	2								1	1	1				1
<b>TENOTOMY.</b>																
a. For talipes	14	3	11	8	4	1	1					3	10	1		
b. For torticollis	4	3	1	4								1	3			
<b>REMOVAL OF TUMOURS.</b>																
Molluscum contagiosum	1	1							1							1
Epulis	2	2			1		1		1				2			
Warts	3	2	1			2	1						3			
Polypus	2	1	1						1		1			2		
Papilloma	3	3								1	2	2			1	
Tumour of bursa patellæ	4	4						2	1	1	4					
Fibroma	2	2						1	1				1		1	
Adenoma	4	4						2	2			3			1	
Exostoses	1	1			1		1						1			
Enchondroma	1	1											1			
Angioma	2	2	1						1				1	1		
Lipoma	3	1	2						1	1	1	3				

TABLE III—*continued.*

SURGICAL OPERATIONS.	Total.	Sex.		Ages.								Result.				
		M.	F.	5	10	15	20	30	40	50	60	+	C.	R.	U.	D.
<b>REMOVAL OF TUMOURS (continued).</b>																
Cystic tumours . . .	4	1	3	1	1	...	2	...	...	...	...	3	...	1		
Sarcoma . . . .	4	1	3	...	...	1	1	1	...	1	...	3	1	...		
Carcinoma . . . .	13	2	11	...	...	...	...	2	6	4	1	8	...	1	4	
Epithelioma . . . .	5	5	...	...	...	...	1	2	1	1	4	1	...	...		
<b>Totals</b> . . . .	<b>291</b>	<b>169</b>	<b>122</b>	<b>45</b>	<b>19</b>	<b>28</b>	<b>29</b>	<b>45</b>	<b>45</b>	<b>32</b>	<b>31</b>	<b>17</b>	<b>157</b>	<b>69</b>	<b>10</b>	<b>65</b>

## RICHARD GULLETT WHITFIELD.

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THE death of Richard Gullett Whitfield constitutes an epoch in the history of St. Thomas's, if not in that of the large endowed hospitals of London. He was the last of a series of officials who, though now improved away, represented in their own persons the traditional glories of their hospitals, and wielded probably more real power than surgeons, physicians, or even treasurers. They were apothecaries, which means that they occupied the finest houses, after the treasurers', within the hospital walls, that they dispensed more lavish hospitality than any other hospital official, that the drug and wine departments were their immediate charge, that they were the resident medical officers, and as such had the entire medical care of the patients in the absence of the physicians and surgeons, that the ventilation and general sanitary condition of the wards were under their supervision, that the sisters and nurses, and other subordinate officials, had to obey their orders, that even the dressers were in some degree under their governance, and that they shared the pupils' fees. They were medical secretaries, which implies that they attended all meetings of the medical officers and lecturers, and kept their minutes, that they had votes equally with other members of the school, that they were the treasurers of the school fund, that they were the depositories and expounders of all unwritten laws and regulations, and that the general management of the school, of the school-buildings, and of the *employés* was in their grasp. For school and hospital alike they were permanent grand viziers, and it was largely if not mainly through them that treasurers derived their know-

ledge of hospital and school affairs, and through the information thus acquired, if not through them, that they acted for good or evil.

The post of apothecary to St. Thomas's Hospital had been hereditary in Mr. Whitfield's family. His grandfather, who was of an old Shropshire family, was appointed in 1752, and continued in office until 1800, dying at the age of seventy-six in the course of the following year. His father succeeded to the apothecaryship in 1800, and held it until 1832, dying, aged sixty-seven, five years later. On his retirement the subject of the present memoir received the appointment.

Mr. Whitfield was born on the 31st January, 1801, was sent to Eton when about nine years old, and continued there until he went to St. John's College, Oxford, where he matriculated in 1818 or 1819. It was then intended that he should take his degree and become a physician. But he had not been at college more than a term or two when he was suddenly recalled to London and entered as a student at St. Thomas's Hospital. It was at this time that the united schools of St. Thomas's and Guy's and the rival school of St. Bartholomew's were at the zenith of their reputation, and Mr. Whitfield, like many others, studied at both. In 1822, when twenty-one years of age, he became a licentiate of the Society of Apothecaries, and in the same year was appointed assistant to his father. He became a member of the Society of Apothecaries in 1824, married in 1828, and was elected apothecary in 1832, and medical secretary in the course of the next year. He continued apothecary down to the removal of the hospital to its present site in the year 1871, then retiring on a pension, and medical secretary to within a month or six weeks of his death, which took place on February 20th of the present year, at the ripe age of seventy-six.

No man was ever more thoroughly identified with an institution than Mr. Whitfield with St. Thomas's Hospital; he loved it with an almost idolatrous love; its success was his success, its failures his failures, and he strove heart and soul through a long life to further in every way what he considered to be its best interests. The separation between the united schools of St. Thomas's and Guy's occurred while he

was assistant to his father—at a time, therefore, when probably he was compelled to be a mere passive spectator of a crisis which he deplored. But no other event of any importance to the hospital or school took place in which his influence was not more or less conspicuous. It was in great measure through his ascendancy over the late treasurer, Mr. Baggallay, that the early hour of visit to the hospital wards, which has now been continued for nearly twenty years, was forced upon the medical staff. When, a few years later, the question of the removal of the hospital from its historic site in the Borough came on for decision, he advocated with characteristic and persistent vigour its translation into the country, with the retention of only a small supplementary hospital or receiving house in London. But when he found his hopes in this direction frustrated, he co-operated loyally with his colleagues and the governors in securing the site at Stangate, and he was largely, if indirectly, concerned in determining the construction of the hospital on the pavilion principle.

He was not great as a physician, nor were his attainments in chemistry or pharmacology of a high order; still he was a competent and zealous medical officer, and an excellent head of the pharmaceutical department of the hospital, and in both capacities commanded the respect of his nominal superiors. It was mainly, however, as an organiser and administrator that he excelled. Under his immediate supervision and direction the ventilation and general sanitary condition of the hospital were never for a moment neglected; he gave much attention to the improvement of the diet and the promotion of the comfort of the patients; the question of out-patient hospital relief was one whereon he had definite and enlightened views, which he embodied in a pamphlet; and the improvement of the nurses and of the nursing which has been so thoroughly effected in St. Thomas's Hospital during the last twenty or five-and-twenty years was due in no small degree to his persistent advocacy and efforts. Further, it was largely owing to him that the Nightingale training-school for nurses was successfully established and carried on. At its institution in 1860 he was appointed medical instructor, and down to his retirement from the post in 1872 he taught the

probationers by lectures and examinations, and conducted the establishment jointly with the superintendent, Mrs. Wardroper. It might be assumed from what has been said that he took little interest in the practice of medicine: the assumption would be so far true that he took little interest in individual cases of disease and in their scientific study, but he took a real interest in the diseases originating in hospitals, and in epidemics during their epidemic prevalence; and few who were at the hospital during the autumn of 1854 will forget the unselfish devotion with which, during that anxious period, he tended almost unaided the numerous cholera patients that were admitted within the hospital walls.

But Mr. Whitfield had other tastes. He was a great lover of nature. His house in London was always bright with choice exotics, and noisy with valuable pet dogs. He was also, from his school-days, when he shot and stuffed his first bird, an ardent and omnivorous collector, and objects accumulated year by year until every room and passage in his house was a museum, and numbers of valuable specimens had to be stowed away in cupboards and lumber-rooms. When the second Alexandra Palace was opened, his treasures, which were then for the first time fully and worthily displayed, completely filled the large room which was devoted to them. The collection, it may be recollected, comprised stuffed animals of all kinds, skeletons, shells, corals, sponges, and numerous other aquatic animals and vegetables, implements of warfare, and innumerable other specimens of interest and rarity; some of them given to him by grateful patients, many by old students who had visited or were settled in foreign countries and knew his tastes. He was a Fellow of the Zoological Society.

Any record of Mr. Whitfield would be unpardonably incomplete which failed to give some description of his personal appearance and manner and of his mental characteristics. He was of middle stature, of strong and compact build, with a fine head, well-formed and expressive features, and florid complexion. Dignity was stamped on every lineament and movement. He was, moreover, nice in his dress, and had an almost feminine love of finery. He had the reputation (which enhanced his importance in the eyes of medical students) of being expert in boxing and other athletic exercises. He was

a man of marked character, and as such made enemies as well as friends. He was bold, determined, and quick-tempered, fond of power, formed his own opinions of things, and acted in accordance with his opinions. But he was, above all things, generous and kind-hearted, lavish of hospitality, and the ever ready benefactor of all who were in need. Many an old student of the old hospital in the Borough will call to mind the pleasures of his hospital table, and many a student, too, will recall with a tear of gratitude the services secretly rendered to him, the money freely given to him, with no thought of repayment, at the moment of peril or of need. The ascendancy which he acquired over the sisters, nurses, and all other hospital officials, from the highest to the lowest, was remarkable. It was due, no doubt, partly to tact, but it was also largely referable to the combination of strength of purpose, good sense, knowledge of his own and of their duties, and genuine kindness, which were manifested and recognised in all his actions. In dealing with women his ascendancy was further aided by his unfailing courtesy to them ; for if a little rough, possibly even somewhat repellent at times, in his intercourse with men, he was always polite and always courteous to the opposite sex.

As age crept over Mr. Whitfield he still retained in a large degree the characteristics of his prime. He was still erect, still dignified, still handsome ; his face was still smooth and ruddy ; and he looked a dozen years younger than he was. His hair was scarcely shot with grey. He was still self-reliant, fond of power and quick of temper, still generous and kind-hearted. But those who knew him only as secretary to the Medical School at Stangate, knew a very different man from him who had been apothecary and medical secretary in the Borough. He was no longer the central figure in St. Thomas's Hospital ; the reins of power which he had so long held had slipped from his grasp ; he was a nonentity within the walls wherein he had once reigned paramount. And even in relation to the school, with which he was still associated, his authority was questioned, and by some of those who were only recently connected with it he was apt to be regarded as impracticable and obstructive. Unfortunately, too, he was becoming infirm, less from the inroads of time than from the

effects of painful illness, and incapable of exercising that efficient supervision which is required of a secretary. But he clung to office and to the semblance of power almost to the last, only sending in his resignation when he lay prostrate with illness on the bed from which he never rose again. He died as he had lived, a genuine, true-hearted English gentleman; mourned by a widow (to whom he had been married for nearly fifty years) and by a numerous family, who were devoted to him; mourned not less sincerely by all those colleagues with whom his official life had been associated, and by numbers of others to whom his kindness of heart or substantial charity had through a long life endeared him. He was followed to his final resting place at Norwood, not only by members of his own family, but by the Treasurer of St. Thomas's Hospital, by the great majority of the medical and surgical staff and lecturers, and by a large assemblage of hospital students, and other friends.

MEDICAL OFFICERS AND LECTURERS  
 OF  
**St. Thomas's Hospital**  
 AND  
**MEDICAL SCHOOL.**

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**SECRETARY.**

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**DEAN OF THE SCHOOL.**

Dr. ORD.

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**LECTURES AND DEMONSTRATIONS.**

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Dr. BRISTOWE } ... ... ... Medicine.  
 Dr. ORD } ... ... ...

Dr. BRISTOWE  
 Dr. MURCHISON } ... ... ... Clinical Medicine.  
 Dr. STONE } ... ... ...  
 Dr. ORD } ... ... ...

Dr. MURCHISON ... ... ... Clinical Medicine, Special Course.

Mr. SYDNEY JONES } ... ... ... Surgery.  
 Mr. MAC CORMAC } ... ... ...

Mr. S. JONES  
 Mr. CROFT } ... ... ... Clinical Surgery.  
 Mr. MAC CORMAC } ... ... ...  
 Mr. MASON } ... ... ...

Dr. PAYNE ... ... ... ... General Pathology.

Mr. MASON } ... ... ... Descriptive Anatomy.  
 Mr. W. W. WAGSTAFFE } ... ... ...

Dr. JOHN HARLEY ... ... ... General Anatomy and Physiology.

Mr. LIEBREICH	...	...	Ophthalmic Surgery.
Dr. BERNAYS	...	...	Chemistry and Practical Chemistry.
Dr. GERVIS	...	...	{ Midwifery, and the Diseases of Women and Children.
Dr. STONE	...	...	Physics and Natural Philosophy.
Dr. STONE	...	...	Materia Medica, and Therapeutics.
Dr. PAYNE	...	...	Forensic Medicine.
Dr. CORY	...	...	
Dr. GREENFIELD	...	...	{ Morbid Anatomy and Practical Pathology.
Mr. A. W. BENNETT	...	...	Botany.
Mr. C. STEWART	...	...	Comparative Anatomy.
Dr. WM. RHYS WILLIAMS	...	...	Mental Diseases.
Dr. A. CARPENTER	...	...	State Medicine.

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#### TEACHERS OF PRACTICAL SUBJECTS AND DEMONSTRATORS.

Dr. T. C. CHARLES	...	...	Practical Physiology.
Dr. BERNAYS	...	...	Practical Chemistry.
Mr. CROFT	...	...	
Mr. MAC KELLAR	...	...	Practical and Manipulative Surgery.
THE ANATOMICAL LECTURERS	...	...	
Dr. REID and ASSISTANTS	...	...	Demonstrations in Anatomy.
Mr. RAINES	...	...	{ Demonstrations in Microscopical Anatomy.
Dr. GREENFIELD	...	...	Demonstrations of Morbid Anatomy.
Dr. T. C. CHARLES	...	...	Demonstrations in Physiology.
			Demonstrations in Practical Physiology.
Dr. PAYNE	...	...	Diseases of the Skin.
Dr. GREENFIELD	...	...	Diseases of the Throat.
Mr. CLUTTON	...	...	Diseases of the Ear.
Mr. J. W. ELLIOTT	...	...	
Mr. W. G. RANGER	...	...	Diseases of the Teeth.

**TIMES OF ATTENDANCE OF THE PHYSICIANS AND SURGEONS IN THE WARDS.**

	Mon.	Tues.	Wed.	Thurs.	Fri.	Sat.
DR. BRISTOWE . . . . .	—	2	—	—	2	—
DR. MURCHISON . . . . .	2	—	—	2	—	—
DR. STONE . . . . .	2	—	—	2	—	—
DR. ORD . . . . .	—	2	—	—	2	—
DR. GERVIS . . . . .	2	—	—	2	—	—
MR. SYDNEY JONES . . . . .	—	2	—	—	2	—
MR. CROFT . . . . .	2	—	—	2	—	—
MR. MAC CORMAC . . . . .	2	—	—	2	—	—
MR. MASON . . . . .	—	2	—	—	2	—
MR. LIEBREICH . . . . .	3	—	—	3	—	—

**TIMES OF ATTENDANCE OF THE ASSISTANT-PHYSICIANS AND ASSISTANT-SURGEONS ON THE OUT-PATIENTS.**

	Mon.	Tues.	Wed.	Thurs.	Fri.	Sat.
DR. HARLEY . . . . .	—	12.30	—	—	12.30	—
DR. PAYNE . . . . .	—	—	12.30	—	—	12.30
DR. GREENFIELD . . . . .	12.30	—	—	12.30	—	—
DR. CORY . . . . .	—	—	12.30	—	—	—
MR. MASON . . . . .	12.30	—	—	12.30	—	—
MR. WAGSTAFFE . . . . .	—	12.30	—	—	12.30	—
MR. MAC KELLAR . . . . .	—	—	12.30	—	—	12.30

**TIMES OF ATTENDANCE ON THE OUT-PATIENT SPECIAL DEPARTMENTS. (See p. 386.)**

	Mon.	Tues.	Wed.	Thurs.	Fri.	Sat.
DR. GERVIS (Diseases of Women)	—	—	—	—	12.30	—
MR. LIEBREICH (Diseases of Eye)	3	3	3	3	3	—
DR. PAYNE (Diseases of Skin)	—	—	—	12.30	—	—
DR. GREENFIELD (Dis. of Throat)	—	12.30	—	—	—	—
MR. CLUTTON (Diseases of Ear)	12.30	—	—	—	—	—
DR. CORY (Diseases of Children)	—	—	—	—	—	12.30
MR. ELLIOTT } (Dis. of Teeth)	—	10	—	—	10	—
MR. RANGER } (Dis. of Teeth)	—	—	—	—	—	—

**DAYS AND HOURS OF ATTENDANCE ON LECTURES  
AND DEMONSTRATIONS.**

WINTER SESSION.	Mon.	Tues.	Wed.	Thurs.	Fri.	Sat.
Descriptive and Surgical Anatomy	9	9	9	9	9	—
Anatomical Demonstrations . . .	10-4	10-4	10-4	10-4	10-4	10-2
Chemistry . . . . .	—	10	—	10	10	—
Physics . . . . .	—	—	—	—	—	12
General Pathology . . . . .	—	4	—	—	—	—
Physiology . . . . .	—	4	4	—	4	—
Clinical Medicine, Special Course . . . . .	—	9	—	—	—	—
Medicine . . . . .	4	—	5	4	—	—
Surgery . . . . .	5 or 5	—	—	5	5	—
Practical and Manipulative Surgery . . . . .	—	—	—	—	—	9
Pathological Anatomy . . . . .	—	—	—	—	4	11½-1½

Demonstrations of Morbid Anatomy 2 p.m. daily.

SUMMER SESSION.	Mon.	Tues.	Wed.	Thurs.	Fri.	Sat.
Materia Medica . . . . .	9	—	9	—	9	—
Forensic Medicine . . . . .	—	9	—	9	—	9
Botany . . . . .	—	10	10	—	—	10
Practical Chemistry . . . . .	10-12	—	—	10-12	10-12	—
Comparative Anatomy . . . . .	12	—	—	12	—	—
Practical Physiology . . . . .	—	11-12½	12½	—	—	11-12½
Mental Diseases . . . . .	—	—	—	—	12	—
Midwifery . . . . .	4	4	—	4	4	—
State Medicine . . . . .	—	—	4	—	—	—
Ophthalmic Surgery . . . . .	5	—	—	—	—	—
Practical and Manipulative Surgery . . . . .	—	5	—	—	5	—

Demonstrations of Morbid Anatomy 2 p.m. daily.

The times of delivery of the Clinical Lectures are arranged, in accordance with other work, in the course of the Session.

The Dissecting Room is open to the Students from 9 a.m. till 6 p.m.

SURGICAL OPERATIONS are performed on Wednesdays and Saturdays, at 1.30 p.m., except in cases of emergency.

*Out-Patients* with Diseases of the Skin are seen by Dr. PAYNE, on Thursdays, at half-past 12 o'clock.

Diseases of Women and Children, occurring amongst *Out-Patients*, are treated on Fridays by Dr. GERVIS, and on Wednesdays by Dr. CORY, at half-past 12 o'clock.

Children under seven years of age are also seen by Dr. CORY, on Saturdays, at half-past 12 o'clock.

Ophthalmic cases are seen as *Out-Patients* by Mr. LIEBREICH, on Mondays and Thursdays, at 4 o'clock; by his Assistant daily; and the Operations are performed on Thursdays, at the same hour.

*Out-Patients* with Diseases of the Ear are seen by Mr. CLUTTON, on Mondays at half-past 12 o'clock; and Diseases of the Throat occurring amongst the *Out-Patients* are treated on Tuesdays by Dr. GREENFIELD, at the same hour.

Instruction in *Dental* Surgery is given by Mr. ELLIOTT and Mr. RANGER, on Tuesdays and Fridays, at 10 o'clock.

Post-mortem Examinations by Dr. GREENFIELD, and Pathological Demonstrations, daily, at 2 o'clock p.m.

The Medical and Surgical *Casualty Out-Patients* are seen by the Resident Assistant-Physician, the Resident Assistant-Surgeon, the House-Physician, House-Surgeons, and Dressers, at from 12 to 1 o'clock daily.

In addition to the Clinical instruction given in the Wards and the *Out-Patients'* Rooms by the Medical and Surgical Officers, Lectures on Clinical Medicine and Surgery are delivered weekly during both the Winter and Summer Sessions by the Physicians and Surgeons to the Hospital, and Special Lectures on Clinical Medicine are delivered once a week by Dr. MURCHISON.

Practical instruction in Vaccination is given by Dr. CORY once a week.

## FEES FOR ATTENDANCE ON THE LECTURES AND ON THE PRACTICE OF THE HOSPITAL.

### PERPETUAL TICKETS,

#### *Admitting to Hospital Practice and Lectures.*

The Fee to Hospital Practice and Lectures may be paid in several ways:

- 1st. One Hundred Pounds paid on entrance;
- 2nd. One Hundred Guineas paid in two moieties, one on entrance, and one at the beginning of the next Session;
- 3rd. Payment by three instalments, viz. of 40 Guineas at the beginning of the first year, 40 Guineas at the beginning of the second year, and 30 Guineas at the beginning of the third year, entitles a Student after payment of the third instalment to an unlimited attendance.

Gentlemen entering at St. Thomas's in the second year of their Studentship pay 40 Guineas for that year; 25 Guineas for the third year; 15 Guineas for the fourth year; and 10 Guineas for each succeeding year, or upon paying £65 on entrance they will receive a Perpetual Ticket. Students entering in their third year pay 30 Guineas; for the next year 15 Guineas; and for every succeeding year 10 Guineas, or one payment of £40 on entrance will entitle them to a Perpetual Ticket.

The Fee for attendance on the general subjects required of Students in Dental Surgery is for the two years £45, or by instalments £40 for the first year, and £10 for the second year.

Regularly qualified Medical Practitioners are admitted to the Hospital practice, and to the Lectures and Library, on payment of a fee of Ten Guineas for unlimited attendance.

All privileges in respect of Hospital attendance are granted subject to the approval of the Governors.

### EXTRA CHARGES.

Students attending the Classes of Practical Chemistry and Practical Physiology are required to pay a fee of One Guinea and a half for each Class, as a contribution towards the cost of instruments supplied and of materials used by them.

Students attending the Class of Practical Physiology should at once provide themselves with Microscopes.

Each Student attending the Class of Morbid Anatomy and Practical Pathology will be required to pay a fee of Half a Guinea for reagents and necessary apparatus.

Students Dissecting pay for the parts they dissect at fixed rates, which are notified in the Library.

Each Student attending the Course of Practical and Manipulative Surgery will be required to pay for the parts of the bodies upon which he may operate, at the same rate as for subjects for dissection.

The Clinical Clerks must provide themselves with a Stethoscope and Registering Clinical Thermometer. The Dressers are required to have a Registering Clinical Thermometer, a Pocket Case of Instruments, and a Case of Silver Catheters.

Each Student wishing to make use of the Library will be required to pay a fee of One Guinea for the whole period of his Studies at the Hospital.

*The different Courses of Lectures, or the Hospital Practice, may also be attended separately on the following terms:*

*For the Medical and Surgical Practice, including the Special Departments.*

Three months . . . . .	8 Gu.	Twelve months . . . . .	24 Gu.
Six ditto . . . . .	14 "	Perpetual . . . . .	40 "
Nine ditto . . . . .	19 "		

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*For Lectures and Demonstrations.*

*1 Course. Perpetual.*

Medicine, Surgery, Physiology, Anatomy, Dissections, Chemistry, each . . . . .	5 Gu.	8 Gu.
Midwifery . . . . .	4 "	6 "
Materia Medica, Botany, Physics, Forensic Medicine, General Pathology, and Comparative Anatomy, each . . . . .	3 "	4 "
Mental Diseases, Ophthalmic Surgery, Dental Surgery, State Medicine, each . . . . .	2 "	3 "
*Practical Chemistry, Practical Surgery, Practical Physiology, Morbid Anatomy, and Practical Pathology, each . . . . .	3 "	

Instruction in Pharmacy and Pharmaceutical Manipulation, to meet the requirements of the Royal Colleges of Physicians and Surgeons, and of the Society of Apothecaries, is given in the Dispensary of the Hospital by the Apothecary, Mr. S. PLOWMAN. The fee for this course of instruction is 5 Guineas for three months.

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## SCHOLARSHIPS, PRIZES, APPOINTMENTS, AND OTHER HONORARY DISTINCTIONS.

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### ENTRANCE SCHOLARSHIPS IN NATURAL SCIENCE.

Two Scholarships, of the value of £60 and £40 respectively, will be awarded during the first week in October, after an examination in Physica, Chemistry, Botany, and Zoology. The Examinations for these Scholarships will be held in the beginning of October each year, the subjects being the same as those for the Preliminary Scientific Examination of the London University, viz., Botany, Zoology, Chemistry (including Practical Chemistry), and Natural Philosophy. Those Students shall be entitled to compete who are duly entered as First Year Students of the Hospital, First Year Students being those registered as such at the Royal College of Surgeons for the Winter Session at the beginning of which the Examination is held. This condition is not intended to exclude Students entering at the Hospital during the previous Summer Session.

### THE WILLIAM TITE SCHOLARSHIP.

This Scholarship, founded by the late SIR WILLIAM TITE, C.B., M.P., F.R.S., and endowed with £1000 Consols, producing £30 per annum, is awarded each year to the Student placed highest in the first class list in the examinations at

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\* These amounts do not include the extra charges in the Practical Courses for Materials, Instruments, &c.

the end of the Winter Session. Preference, in case of equality between Students, is to be given to the son of a medical man, and more particularly of one who has been educated at St. Thomas's Hospital or is in practice in Bath.

#### THE MUSGROVE SCHOLARSHIP.

This Scholarship, founded by **SIR JOHN MUSGROVE, Bart.**, the President of the Hospital, and endowed with £1400 Consols, producing 40 Guineas per annum, is awarded biennially to the Student who shall take the highest place in the first class list in the examinations at the end of the Second Winter Session. It is tenable for two years, provided the holder obtains a place in the first class in the subsequent examinations.

#### A COLLEGE SCHOLARSHIP.

A Scholarship, of the value of 40 Guineas, also tenable for two years, will be given every second year, alternately with the Musgrove Scholarship, and on similar conditions, to the Student placed highest in the first class list, at the end of the Second Winter Examination.

Gentlemen obtaining these Scholarships are not precluded from receiving any of the Prizes awarded at the subsequent periodical examinations.

### PRIZES.

#### THE FOLLOWING SCHOLARSHIPS, PRIZES, AND MEDALS, WILL BE AWARDED DURING THE YEAR 1877—78.

TWO ENTRANCE SCHOLARSHIPS IN NATURAL SCIENCE, OF THE VALUE OF SIXTY AND FORTY POUNDS.

#### FOR FIRST YEAR'S STUDENTS.

##### WINTER. SUMMER.

1st. The William Tite Scholarship	£30	1st. College Prize	£15
2nd. College Prize	£20	2nd. Ditto	£10
3rd. Ditto	£10	3rd. Ditto	£5

#### FOR SECOND YEAR'S STUDENTS.

##### WINTER. SUMMER.

1st. Musgrove Scholarship	£42	1st. College Prize	£15
2nd. College Prize	£20	2nd. Ditto	£10
3rd. Ditto	£10	3rd. Ditto	£5

#### FOR THIRD YEAR'S STUDENTS.

##### WINTER.

1st. College Prize £20. 2nd. Ditto . . £15. 3rd. Ditto . . £10.

Students of each year are classed according to their respective merits in the examinations; and those in the *first* class in each year receive Certificates of Honour and a preference in the selection for Hospital Appointments.

In addition there are awarded—

The Cheseiden Medal, The Mead Medal, The Solly Medal and Prize, The Grainger Testimonial Prize, The Treasurer's Gold Medal.

#### THE CHESEIDEN MEDAL,

founded by the late **George Vaughan, Esq.**, is annually awarded to the Fourth Year's Student who most distinguishes himself in a Practical Examination in Surgery and Surgical Anatomy.

#### THE MEAD MEDAL,

founded by **Mr. and Mrs. Newman Smith**, is awarded annually to a Fourth Year's Student, in respect of a special Practical Examination in Medicine.

## THE SOLLY MEDAL.

together with a Prize in Money, will be awarded biennially. Those Students are eligible to compete who shall be of from three to six years' standing. The award is made for the best series of Reports of Surgical cases coming under the Students' personal observation in the Wards, not, however, to exceed ten in number. Preference is given, merit in other respects being equal, to Reports illustrated by the author's drawings, and short Clinical Remarks must accompany each Report. The next award will be made at the end of 1877—78.

## THE GRAINGER TESTIMONIAL PRIZE,

of the value of £20, is awarded biennially to Third or Fourth Year's Students for the best Physiological Essay, to be illustrated by preparations and dissections. Competitors for this Prize must be Medical Students of St. Thomas's Hospital, and on the day of sending in their Essays, Dissections, and Preparations, shall have completed the Second, but not the Fourth year of their medical studies.

## THE TREASURER'S GOLD MEDAL

for General Proficiency and Good Conduct is awarded to the Student who has passed through the whole of his four years' pupilage in the most meritorious manner.

*Two House Physicians and Two Assistant House Physicians, Two House Surgeons and Two Assistant House Surgeons, and a Resident Accoucheur*, are selected from Gentlemen who have obtained their professional diplomas; they hold office for three or six months. The Assistant House Physicians and House Surgeons are non-resident, but the other Officers, together with the Dressers and Obstetric Clerks, are provided with Rooms and Commons during their period of attendance in the Hospital, free of expense.

An Ophthalmic Clinical Assistant is appointed at a Salary of £50 per annum, who may hold office for twelve months.

*Clinical Clerks and Dressers* to In-Patients are selected to the number of at least 40 respectively each year. They are chosen from amongst the most eligible pupils. *Clinical Clerks and Dressers* for the Out-Patients are also appointed to the number of at least 40 to 50 each year.

All Students have the opportunity afforded them of being engaged in the performance of practical duties in connection with the Medical, Surgical, Obstetrical, Ophthalmic, and Pathological Departments of the Hospital.

*Two Hospital Registrars*, at an annual Salary of £40 each, are appointed in each year. Preference will be given to Gentlemen who have been distinguished for merit, and have completed their studies in the School. The payment of the Registrars is subject to the presentation of a Report upon the Practice of the Hospital, and to such Report being regarded as satisfactory by the Medical Officers to whom it shall have been referred.

Two or more Gentlemen are selected from Students who have completed their Second Winter Session, to act as Assistants in the Dissecting Room. They receive Certificates of Honour according to merit.

Prosecutors are appointed in the early part of the Winter Session, and Prizes are awarded to the best Dissectors at the termination of the Session.

Gentlemen are likewise appointed to act as Assistants to the Demonstrator of Pathological Anatomy in the Post-mortem Room.

Obstetric Clerks are from time to time appointed. Each holds office for a fortnight, and Certificates of Honour are awarded to those Gentlemen who have satisfactorily attended Fifty Maternity cases.

Students have access, with the permission of the Officers under whose superintendence they are placed, to the Museums of Human and Comparative Anatomy and Pathology—of *Materia Medica*—of Botany—and of Chemistry and Mineralogy—and to the Laboratories of Practical Physiology and Practical Chemistry; also to the Library, which contains a large collection of works of reference and modern text-books, on payment of a small fee.

## Distribution of Prizes,

On Wednesday, 5th July, 1876, at Three o'clock, by

DR. J. RISDON BENNETT, F.R.S., PRESIDENT OF THE ROYAL COLLEGE OF PHYSICIANS.

### PRIZES FOR SUMMER SESSION OF 1875.

#### SECOND YEAR'S STUDENTS.

Introduced by Mr. CROFT.

C. E. SHEPPARD, *Kensington* ... ... COLLEGE PRIZE £15, and Certificate of Honour.  
 G. B. LONGSTAFF, *Wandsworth* ... DITTO £10, and Certificate of Honour.  
 F. H. WEEKES, *Southampton* ... DITTO £5, and Certificate of Honour.

#### FIRST YEAR'S STUDENTS.

Introduced by Dr. BERNAYS.

J. SHAW, *Clapham Road* ... ... COLLEGE PRIZE £15, and Certificate of Honour.  
 W. E. WOODMAN, *Camberwell* ... DITTO £10, and Certificate of Honour.  
 H. CASTLE, *Newport, Isle of Wight* ... DITTO £5, and Certificate of Honour.  
 S. J. TAYLOR, *Grantham* ... ... Certificate of Honour.  
 S. A. CRICK, *Cosby Hill, Leicestershire* Certificate of Honour.

### PRIZES FOR WINTER SESSION OF 1875-76.

#### ENTRANCE SCIENCE SCHOLARSHIPS.

Students introduced by Dr. STONE.

H. A. H. FENTON, *Westminster* ... Scholarship £60.  
 T. D. SAVILL, *Brixton* ... ... Scholarship £40.

#### FIRST YEAR'S STUDENTS.

Introduced by Dr. ORD.

T. D. SAVILL, *Brixton* ... ... WM. TITE SCHOLARSHIP, and Certificate of Honour.  
 A. NEWSHOLME, *Bradford* ... ... COLLEGE PRIZE £20, and Certificate of Honour.  
 TAKAKI KANEHIRO, *Kasumigaseki Tokai, Japan* ... ... DITTO £10, and Certificate of Honour.  
 C. A. BALLANCE, *Lower Clapton* ... Certificate of Honour.  
 A. PURKISS, *Kensington* ... ... Certificate of Honour.  
 HO KAI, *Hong Kong, China* ... ... Certificate of Honour.

### SECOND YEAR'S STUDENTS.

Introduced by Mr. MAC CORMAC.

S. J. TAYLOR, *Grantham* ... ... ... MUSGROVE SCHOLARSHIP, 40 Gu., and Certificate of Honour.

J. SHAW, *Clapham Road* ... ... ... COLLEGE PRIZE £20, and Certificate of Honour.

### THIRD YEAR'S STUDENTS.

Introduced by Mr. SYDNEY JONES.

G. B. LONGSTAFF, *Wandsworth* ... ... ... COLLEGE PRIZE £20, and Certificate of Honour.

C. E. SHEPPARD, *Kensington* ... ... ... DITTO £15, and Certificate of Honour.

F. H. WEEKES, *Southampton* ... ... ... DITTO £10, and Certificate of Honour.

E. H. D. GIMLETT, *Southsea* ... ... ... Certificate of Honour.

F. W. GILES, *Henley-on-Thames* ... ... ... Certificate of Honour.

### PHYSICAL SOCIETY'S PRIZES.

Students introduced by Mr. WAGSTAFFE.

E. H. JACOB, *Winchester* ... ... ... SOCIETY'S THIRD YEAR'S PRIZE, and Certificate of Honour.

C. E. SHEPPARD, *Kensington* ... ... ... SOCIETY'S SECOND YEAR'S PRIZE, and Certificate of Honour.

D. S. DAVIES, *Bristol* ... ... ... SOCIETY'S FIRST YEAR'S PRIZE, and Certificate of Honour.

### PROSECTORS.

Students introduced by Mr. F. MASON.

S. A. CRICK, *Cosby Hill, Leicestershire* PRIZE, and Certificate of Honour.

R. H. E. KNAGGS, *Trinidad, West Indies* ... ... ... PRIZE, and Certificate of Honour.

### RESIDENT ACCOUCHEURS.

Introduced by Dr. GERVIS.

WALTER EDMUND, *St. John's Wood* Certificate of Honour.

S. W. J. JOSEPH, *St. Leonard's* ... ... ... Certificate of Honour.

G. F. ROSSITER, *Taunton* ... ... ... Certificate of Honour.

C. C. SMITH, *Redditch* ... ... ... Certificate of Honour.

### HOUSE PHYSICIANS.

Students introduced by Dr. BRISTOWE.

C. H. NEWBY, *London* ... ... ... Certificate of Honour.

G. F. ROSSITER, *Taunton* ... ... ... Certificate of Honour.

WALTER EDMUND, *St. John's Wood* Certificate of Honour.

H. P. POTTER, *Denmark Hill* ... ... ... Certificate of Honour.

S. W. J. JOSEPH, *St. Leonard's* ... ... ... Certificate of Honour.

### HOUSE SURGEONS.

Introduced by Mr. SIMON.

H. P. POTTER, *Denmark Hill* ... ... ... Certificate of Honour.

H. H. CLUTTON, *Witham* ... ... ... Certificate of Honour.

C. H. NEWBY, *London* ... ... ... Certificate of Honour.







